S. Hrg. 100-834, Pt. 1

CAUSES AND CONSEQUENCES OF ALCOHOL ABUSE

HEARINGS

BEFORE THE

COMMITTEE ON GOVERNMENTAL AFFAIRS UNITED STATES SENATE

ONE HUNDREDTH CONGRESS

SECOND SESSION

PART 1

OVERVIEW OF FEDERAL ACTIVITIES ON ALCOHOL ABUSE AND ALCOHOLISM
MAY 25, 1988

ALCOHOLISM: BIOLOGICAL AND ENVIRONMENTAL DETERMINANTS
MAY 26, 1988

Printed for the use of the Committee on Governmental Affairs

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OVERVIEW OF FEDERAL ACTIVITIES ON ALCOHOL ABUSE AND ALCOHOLISM

WEDNESDAY, MAY 25, 1988

U.S. SENATE,
COMMITTEE ON GOVERNMENTAL AFFAIRS,
Washington, DC.

The Committee met, pursuant to notice, at 10:30 a.m., in room SD-342, Dirksen Senate Office Building, Hon. John Glenn, Chairman of the Committee, presiding.

Present: Senators Glenn, Sasser, and Roth.

OPENING STATEMENT OF CHAIRMAN GLENN

Chairman GLENN. The hearings will be in order.

Good morning and welcome to the first of four hearings that the Governmental Affairs Committee will be holding on the causes and consequences of alcohol abuse and alcoholism in this country. This Committee has jurisdiction over and responsibility for intergovernmental relations and the cooperation between and relationship of Federal and State programs for various purposes.

In addition, we also have oversight responsibility for the economy and efficiency of all agencies of the United States Government. Therefore, what Federal and State units of government do, or fail to do, in concert with one another on health-related matters is of concern to this Committee.

Earlier this year this Committee looked into nutrition research and monitoring activities carried out by Federal agencies with different missions. In early June we will be examining Federal and State activities concerning the AIDS epidemic.

Beginning today this Committee will be devoting four hearing days, and possibly more later in the year if we can, and into next year. We do not plan to make this just a series of four. Since it is a continuing problem, we are going to have continuing attention on it. The Committee will be devoting four hearing days as of now to an examination of the causes and consequences of alcohol abuse and alcoholism in this country.

There is much activity and concern today about the problem of illegal drug use in America; hard drugs. The Congress is so exercised about this problem it is about to enact legislation giving the military broader powers to deal with law enforcement and drug interdiction than it has ever had in the whole history of this country.

Senator Byrd asked me to serve with several other Senators on a small task force to look into our whole approach to illegal drug use. Almost all of the money appropriated for fighting alcohol and drug abuse goes toward the eradication of illegal drugs in the United States.

For example, the fiscal year 1987 appropriation for the Anti-Drug Abuse Act of 1986 was $1.67 billion. Of that amount, $1.132 billion was allocated for illicit drug enforcement activities of which almost one-third of the total budget—$546 million—was spent on the acquisition, maintenance and operation of military hardware. The remaining monies—$532 million—which were to be spent on alcohol and drug abuse prevention, education and rehabilitation services mainly went to drug abuse prevention activities.

Now, we are forgetting in our war on drugs that the use of alcohol, which is a legal drug—it's classed as a drug—is exacting a heavy toll on our society. It has been reported by the Department of Health and Human Services that alcohol is a factor in nearly 50 percent of all murders, accidental deaths and suicides in this country. In all, an estimated 100,000 to 200,000 lives are claimed by alcohol each year—each year! That's an estimated 25 times more than are claimed by all the illegal drugs combined.

The total direct and indirect economic costs associated with alcohol abuse in 1983—which is one of the last years we have good figures for, as I understand it—were estimated to be almost $120 billion—I'm sure that would be up to $150–160 or more billion now—of which 69 percent was due to lost employment and reduced productivity. In comparison, the economic cost to society from drug abuse amounted to $59 billion.

Almost 10 million Americans are classified as alcoholics—those persons who experience memory loss, an inability to stop drinking until intoxicated, and binge drinking. Eight million more Americans are considered problem drinkers. Consumption of beverage alcohol has been associated with an increased risk of developing liver diseases, hypertension, cardiovascular disease and certain forms of cancer.

An estimated 5,000 babies are born each year with fetal alcohol syndrome, a constellation of structural and functional birth defects attributed to in utero exposure to alcohol. As many as 50,000 babies may be born each year with a milder form of alcohol-related birth abnormalities.

Moreover, children of alcoholic or alcohol dependent parents have a four-fold—four times greater—risk of becoming alcoholics themselves when compared to children of non-alcoholic parents.

I will give some additional figures on this, too. It is the most widely used drug among American youth. In 1986 one out of every three adolescents between 14 and 17 experienced poor school performance, trouble with parents or involvement with law enforcement personnel because of alcohol. One in four American homes have been afflicted by an alcohol-related family problem. Recent surveys indicate the first drinking experience today usually occurs around the age of 12.

Thirty-six percent of fourth grade respondents to a 1987 poll reported other kids their age pushed them to drink beer, wine or liquor. That's the fourth grade. In 1986, 52.1 percent of highway deaths were alcohol-related. That's about half of the 45,000 to 50,000 deaths we have on the highway each year; half of them alcohol related.

We get excited about things like the Vietnam war. Over a 10-year period we had 57,000 people killed in the Vietnam war. We kill nearly that many every 2 years just from alcohol on the highways. If we want to know what the impact can be, go to Kentucky. Go to Carrollton, Kentucky and talk to some of the people there. They had a drunk driver coming down the interstate on the wrong side of the road. And two of every five people in the U.S. will be in an alcohol-related crash in their lifetimes. Those are just a few little figures to bring it home as to how enormous this problem is.

Today's hearing and the hearing tomorrow will explore the causes of alcohol dependency and alcoholism among susceptible groups of people and what state and Federal governments have done and could do or are not ding to reduce alcohol-related disease processes that will rqeuire imaginative solutions. We hope

In light of the recent Supreme Court decision of *Traynor* vs. *Turnage,* we would like to solicit our witnesses' opinions on whether they believe that alcoholism is a medical disease or merely willful misconduct. The intent of these hearings is not to initiate a call for a return to prohibition, nor do we intend to suggest or imply that all persons who consume alcoholic beverage products are equally at risk of becoming addicted to alcohol.

Obviously, alcoholism and alcohol dependency are complex disease processes that will require imaginative solutions and we hope that these hearings will shed some light on what causes alcohol addiction and alcoholism, on what we are as a Nation doing to treat alcohol addiction once it occurs, and what we can do to prevent alcohol addiction from occurring in the first place.

We will seek to apply what we learn about the causes and consequences of alcoholism and our societal responses to this massive problem to our more general problem of drug abuse prevention and treatment.

Senator Roth.

Senator ROTH. Thank you, Mr. Chairman.

OPENING STATEMENT OF SENATOR ROTH

I congratulate you for this series of hearings being held on alcohol abuse and alcoholism. I particularly want to congratulate you, Mr. Secretary, for the number of initiatives that you have been taking to help deal with this most serious problem.

Like the Chairman, we would like some answers. I suspect that there are no simple answers, yet here on the Hill too often we try to find them. Nevertheless, alcohol abuse and alcoholism are difficult problems that we need more knowledge about, and we need more effective action on the part of Government at every level.

Our Chairman, Mr. Secretary, mentioned that alcohol is a legal drug and much of our attention is focussed on dealing with illegal drugs. I have always been bothered, although it may be inappropriate, with trying to lump the problems of alcoholism together with the use of illegal drugs. This is an area where I would be very much interested in your expertise.

Should we be treating the two problems—the problem of alcoholism, the problem of illegal drugs—together? Or would it be better that they be dealt with separately? I am concerned that there is a great deal of discussion, right now for the first time, by prominent individuals regarding the desirability of legalizing what we call "hard" drugs.

I wonder what our experience with alcohol—what light it can shed as to whether drug legalization would be a desirable course of action. I must confess that I have very, very serious reservations that this is the appropriate answer. Perhaps legalization might have some favorable impact on the reduction of crime. I do not know. But what impact would such a law have on the use of hard drugs, illegal drugs, by the public at large?

Certainly the legalization of alcohol has not eliminated the abuse of alcohol. I do not know whether alcohol legalization sheds any light on what we could expect if we legalized drugs or not. But anyway, I would be interested in your comments in this area.

Mr. Chairman, I do have another hearing so I may not be able to stay for the entire meeting, but I again want to thank you for initiating this series of hearings.

Chairman GLENN. Thank you, Senator Roth, very much. I know you have been interested in these hearings. You and your staff have worked hard to help them get set up, also.

I would just indicate, before we get to our witnesses here—and I do not want to take long on this—that I think the problem of alcoholism is being recognized more now as a major national problem. Just in my background material the staff had put together here, there is a reprint out of *Time* magazine from November of last year: "This is one of 18 million Americans who have a drinking problem but there is hope; science is discovering the causes of alcoholism and new ways are emerging to cope." A very good article in this.[1]

Newsweek: "Alcohol and the family; growing up with alcoholic parents can leave scars for life." And a lot of information in that one.[2]

Another one out of *U.S. News & World Report* from November of last year: "Coming To Grips With Alcoholism." [3]

I'm going to ask staff right now—and I had not thought of doing this before—I would like to get about 100 copies, reprints, or something of that of each of these tomorrow and we will have them available before the hearing tomorrow for the press and anyone else that wants them.

These are a very good compilation of facts and figures that I think do a great service when our national news publications put out things like this and help make us aware of the problem that we have. We can determine the exact number, but I would like to get them available and have them available for press and anyone else that comes to the hearing tomorrow so they will have some of this same background information, too.

We are very fortunate today to have with us today some experts in this field. The person to lead off our hearing is Dr. Otis Bowen. We are proud to have him here this morning. He has been a spokesman in this area, and has been working tirelessly in this field.

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[1] See p. 350.
[2] See p. 359.
[3] See p. 366.

Dr. Bowen, is Secretary of the Department of Health and Human Services. He is accompanied by Mr. Thomas Burke, Chief of Staff, and Mr. Robert Trachtenberg, Deputy Administrator for the Alcohol, Drug Abuse and Mental Health Administration, and Dr. Enoch Gordis, Director, National Institute of Alcohol Abuse and Alcoholism.

All of them are part of our first panel which we had titled, Overview of Federal Alcohol Abuse Programs.

Dr. Bowen, we welcome you this morning. We are glad to have you with us and we would welcome your statement.

TESTIMONY OF OTIS R. BOWEN, M.D., SECRETARY, DEPARTMENT OF HEALTH AND HUMAN SERVICES,[1] ACCOMPANIED BY THOMAS R. BURKE, CHIEF OF STAFF,[2] ROBERT TRACHTENBERG, DEPUTY ADMINISTRATOR, ALCOHOL, DRUG ABUSE, AND MENTAL HEALTH ADMINISTRATION, AND ENOCH GORDIS, M.D., DIRECTOR, NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM[3]

Secretary BOWEN. Thank you, Chairman Glenn and Senator Roth.

I appreciate the opportunity to testify this morning on the problems of alcohol abuse and alcoholism in our Nation today. As you have mentioned, with me today on my right is Tom Burke, Chief of Staff with the Department and on my left is Bob Trachtenberg, the Deputy Administrator of the Alcohol, Drug Abuse and Mental Health Administration, and Dr. Enoch Gordis, the Director of the National Institute on Alcohol Abuse and Alcoholism.

Later in this hearing Dr. Gordis will provide a comprehensive description of our alcohol research programs for you and Mr. Burke will elaborate on our progress with my initiative.

I have reduced the length of my remarks according to your request, but have submitted the full text to you.

Chairman GLENN. The entire statements of all of you will be included in the record. If you choose to abridge them somewhat, that's fine. That will give us more time for discussion. Thank you.

Secretary BOWEN. First, I want to communicate to you my own deep concern about the tragic—and needless—toll taken by alcohol abuse and alcoholism on our society and its people. And then I would like to outline for you the major initiative that I have undertaken.

As a physician in Indiana, I saw firsthand many of themedical problems caused by alcohol abuse and alcoholism. I witnessed the suffering caused to both patients and their families, a wide range of health and social problems—such as birth defects, accidents, mental illness, other drug use, family violence, teen suicide and homelessness.

And there is the economic factor. Alcoholism and alcohol related problems are estimated to cost this country more than $117 billion a year in everything from health care bills to loss of productivity. If we can tackle successfully the alcohol problem in society today, many of these other problems we face would be dealt a considerable blow, if not eradicated entirely.

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[1] See p. 87 for Secretary Bowen's prepared statement.

[2] See p. 109 for Mr. Burke's prepared statement.

[3] See p. 122 for Dr. Gordis' prepared statement.

Early last year I set out to build on what has already been done in the alcohol abuse and alcoholism fields. Working together with health professionals and other interested people outside the field, both in the public and private sectors, we can make the problems of alcohol abuse and the disease of alcoholism highly visible. This should be an urgent priority for the American public. It certainly is at HHS.

I became convinced that the high visibility of my office could be used to create a national agenda to prevent and reduce alcohol related problems. It was just a year ago today that I convened a distinguished panel of experts to advise me on what was currently being done and what I, as Secretary, could do to stimulate further action.

I received from this panel a frank and unedited report which detailed their findings in five major areas of study: Research and Practice; Education, Information and Media; Treatment; Public Policy and Legislation; and Federal Leadership.

Working from the exhaustive report, I formulated a comprehensive national initiative, which I announced last November at our first ever Secretary's National Conference on Alcohol Abuse and Alcoholism.

I believed then, and believe even more so today, that we need to develop a public-private partnership committed to raising public awareness. This national initiative on alcohol abuse and alcoholism is a multi-pronged effort. It includes components for: better coordination of HHS alcohol related activities; speedy transfer of research findings to the clinical practitioner; fostering research into prevention efforts; improving health professional education; mobilizing Federal efforts to encourage small businesses to participate in employee assistance programs; a public-private sector national media and information campaign; targeting new efforts aimed at identified high-risk populations such as women, specific ethnic groups and children of alcoholics; and the establishment of a privately run National Citizen's Commission.

In trying to design a comprehensive strategy we recognized two areas in which progress could be made to help health care providers to intervene in alcohol abuse and alcoholism: identification of the problem; and barriers to treatment, be it patient resistance, lack of clinical training, or scarcity of new research findings in the field.

My colleagues in the medical community have a unique opportunity to educate themselves and their patients about alcohol abuse. For example, they can take advantage of a joint Health Resources and Services Administration and Office of Substance Abuse Prevention project to improve the ability of primary care providers in community health centers and maternal and child health programs to diagnose, refer and treat alcohol related medical problems and alcoholism.

But let us not underestimate the barriers that physicians and other providers have in treatment. For one, patients themselves may resist treatment, either because they have not admitted their problems or because of stigma attached. But another reason may be that training in alcoholism diagnosis, intervention and treatment is only a very limited part of most health professions education programs—medical schools and residency programs included. Of physicians polled by the "Journal of the American Medical Association" back in 1982, 71 percent said they felt ill-prepared for or ambivalent about making alcohol diagnoses on patients.

My initiative includes a recognition of these problems of medical education and intervention—and we have taken several actions to help. One is the recent ADAMHA program focusing on primary care providers through the development of curriculum models.

Another example of our efforts is the ongoing discussions between NIAAA and medical and nursing organizations. We are urging these groups to play an advocacy role in increasing alcohol training opportunities, including both undergraduate and graduate medical education. And of note are Dr. Gordis' continuing meetings with major health foundations to encourage increased private sector efforts to improve clinical training.

A new "quick response" publication that is called "Alcohol Alert" has as its purpose to get new research findings quickly to a broad array of clinical personnel who can put them into action.

Let me now turn to some of our efforts directed at the public, and in particular at special population groups. Alcohol abuse and alcoholism are inextricably linked as cofactors to numerous other public health problems—cancer, diabetes, hypertension, child abuse, family violence, homicides and infant mortality, among others.

As you can see from the packet that we have provided for all of you, we have engaged in an extensive campaign to GET THE WORD OUT. This is the key, whether you are dealing with the Hispanic population, Indians, youngsters, homeless individuals, children of alcoholics, or any other group.

Health messages can play a vital role in increasing public awareness. It has worked well in our "Healthy Mothers, Healthy Babies" campaign. We are also exploring initiatives with the media experts. For example, my staff has met with the Advertising Council in New York to explore an innovative new television campaign on alcoholism.

We have agreed to work with the American Broadcasting Company on a major prime-time program devoted to a better understanding of alcohol abuse and alcoholism. And the National Broadcasting Company is exploring the idea of making alcoholism and alcohol abuse the centerpiece of their January public service campaigns.

Since our time this morning is limited, I would like to just focus briefly on a few groups—children, the elderly and pregnant women.

A statistic I was shocked to learn is that an estimated 4.6 million adolescents in America, or 3 out of every 10 teenagers, experience serious alcohol problems each year. Equally shocking is the fact that nearly 9 out of 10 teenage auto accidents involve the use of alcohol. And it is no secret that alcohol increases the risk for a range of other problem behaviors, for example, suicide and homicide.

And it has been shown that both alcohol and tobacco are "gateway" drugs. Children and alcoholics who use these generally are at greater risk for using other forms of drugs. Teen pregnancy can be alcohol related. Studies I have seen indicate that in almost half of all teen pregnancies, one or both of the teenagers involved had been drinking alcohol at the time of conception. It is not just high school kids. Younger kids are having problems with alcohol, too.

We are using many weapons to fight this problem: aggressive outreach, public-private cooperative efforts and, importantly, education.

The Office of Substance Abuse Prevention, or OSAP, is carrying out an aggressive demand reduction prevention program targeted to America's young people, and in particular, high risk youth and their families. In the fall of 1987, OSAP awarded 131 demonstration grants, for the development of model, community-based alcohol and other drug prevention programs for high risk youth.

We are working with Hanna Barbera, the animation company, to put together a series of public service announcements on alcoholism and alcohol related problems aimed at children and scheduled for airing on Saturday mornings.

We are going to continue our "Be Smart, Don't Start" campaign aimed at 8 to 12 year olds, and we are going to add a version in Spanish. We have just filmed a new television spot aimed at young people which will feature the actor, Michael J. Fox.

I have also enlisted the Surgeon General, Dr. C. Everett Koop. Recently Dr. Koop held a workshop on health promotion and aging. The first 36 recommendations concerned alcohol issues and the elderly. I'm committed to making sure that these recommendations are being carried out.

Let me turn to another issue—fetal alcohol syndrome, or FAS. I do not have to mention how needless that suffering is. FAS is one of the top three causes of birth defects in the United States today. Babies born with FAS are more likely to be of low birth weight, a major risk factor for infant mortality. As a member of the National Commission to Prevent Infant Mortality, I have tried to bring to the forefront this shocking relationship between drinking during pregnancy and infant death.

The saddest part is, it is totally preventable. If the mothers had stopped drinking when they became pregnant, the babies could well have been born healthy. And that's why education is so important. Some of the materials we have developed to target pregnant women are included in your packet, and here is one of them.

I would be remiss if I did not highlight the important work of the National Citizens Commission. Mrs. Betty Ford has agreed to serve with me as honorary co-chairperson. Robert Raclin, our former Deputy Under Secretary, is the chair, and Dr. Koop is an honorary trustee. The goals of the Commission are to focus public awareness on the problem, stimulate new action in the areas of research, prevention and teaching, and advance the alcohol initiative.

This fall we will be holding our second national conference on alcohol abuse and alcoholism with the National Citizens Commission as our sponsor. We will take a closer look at how alcohol abuse and alcoholism affect child abuse, teen pregnancy, homelessness, school dropout rates, physical fitness and AIDS, as well as such issues as alcohol warning labels and alcohol taxation.

I believe we can elicit change in how people view these problems if we are accurate in our representations and practical in our approach. We need to reach people, to talk with them in words they can understand and to reach them in familiar settings. In particular, we need to reach our children, and teach them to say no to alcohol and other drugs.

Our national agenda is ambitious but I believe it is achievable. Thank you.

Chairman GLENN. Thank you, Doctor, very much. Some of those figures really jump out at you. Three out of every 10 teens will be involved with alcohol problems each year; is that correct?

Secretary BOWEN. Correct.

Chairman GLENN. And nine-tenths of the auto accidents of teens are alcohol related?

Secretary BOWEN. That is correct.

Chairman GLENN. Nine-tenths. Almost all of them.

Do you have the National Conference schedule yet for this fall? Do you know when it will be and where it will be held?

Secretary BOWEN. It will be held in San Diego in November. I do not recall the exact dates. It is in November sometime in San Diego.

Chairman GLENN. Well, we would like to keep in touch with you on that.

Secretary BOWEN. He says October 31.

Chairman GLENN. October 31, Halloween. All right. That is easy to remember. Thank you.

Mr. Burke, do you have a statement?

Mr. BURKE. Yes, sir, I do.

Chairman GLENN. Fine. Go ahead.

Mr. BURKE. Chairman Glenn, Senator Roth, I am pleased to have this opportunity to amplify on the statements of Dr. Bowen indicating the urgent priority of alcohol abuse and alcoholism in our Department. I have summarized my statement quite a bit so I will proceed.

As an economist, I view the effects of alcohol abuse and alcoholism on our Nation's economy to be as damaging as Dr. Bowen has indicated they are to our Nation's health. Each year we do not act, these problems, which are preventable and treatable, steal valuable resources from our national treasury.

Dr. Bowen has told you that the estimated cost of alcohol abuse and alcoholism was $117 billion in 1983. This is projected to go to $136 billion by 1990, just 18 months away, and to $150 billion by the year 1995. That is about the magnitude of our current deficit.

This puts a drain on the Federal Treasury, for example, through increased Medicare, Medicaid and other health care payments, family support payments and programs, and funds for the homeless. Of the $117 billion, the greatest portion—$92.8 billion—comes from reduced worker productivity and lost employment.

This represents products, goods and services never produced and never delivered. These hidden costs represent major economic losses caused by reduced productivity, premature loss of life, employment lost by victims of alcohol related motor vehicle accidents and the added cost of incarceration of criminals.

The economic costs do not even begin to account for the premature loss of loved ones, such as the recent tragedy we witnessed last week in Kentucky that the Chairman alluded to in his opening remarks.

Alcohol related problems are estimated to be directly responsible for a sizeable portion of the Nation's health care costs, an estimated $14.9 billion annually. The enormity of this figure is directly attributable to the many severe medical problems caused by alcohol abuse and alcoholism. Based on a solid body of research, we know that families of alcohol abusers and alcoholics utilize health care services at twice the rate of those who do not have an alcoholic family member.

We also know there is a reduction in these expenses following treatment of the disease for both the family members and the alcoholic. It is amazing that in view of the escalating health care costs, we continue to treat the medical consequences of alcohol abuse and alcoholism rather than its cause. Unfortunately, as a nation we have not yet reached the point where public concern over the damages caused by alcohol and public pressure to prevent and deal with it approach the level of concern expressed about other major illnesses or the abuse of illegal drugs. As the Secretary alluded to, from 25 to 30 percent, and maybe even half of our hospital admissions are said to be in some way or another alcohol related.

An area in which we have sent mixed signals——

Senator ROTH. I am sorry, but I did not hear you. Half of the what?

Mr. BURKE. It has been estimated that 25 to 30 percent and perhaps as high as one-half of the hospital admissions in the United States—the American Hospital Association studied this in 1983—are alcohol abuse related.

We require health warning labels on all sorts of consumer products, tobacco, certain foods and other legal drugs, yet we do not when it comes to products containing the drug alcohol. What does this tell the American public about the harmful consequences of alcohol abuse?

As you know, our recent HHS report to Congress concluded that health warning labels do have the potential to affect consumer behavior, particularly in combination with other educational efforts. The Treasury Department, which has regulatory jurisdiction in this matter, has been required by the Senate Appropriations Committee Treasury Postal Service Subcommittee to undertake a somewhat similar study to what we conducted, and I believe the results of this study are due later this year. Part of this study, I am told, will include a nationwide public opinion survey on the subject of warning labels, and we anxiously await the results of the Treasury study.

For business and industry, alcohol abuse and alcoholism are major factors in lost productivity. We estimated that in the Federal work force alone approximately 8 percent of the 3 million individuals suffer from alcoholism. This year, for the first time, 3 million Federal employee's have access to alcoholism treatment through expanded insurance coverage in employee benefit plans. The benefits of employee assistance programs are many—substantially fewer sick days, fewer work related accidents, improved productivity and lower health care costs. And lower health care costs, of course, increases our international competitiveness.

As a nation, we have a percentage of our GNP devoted to health care that is more than twice what it is in the other industrialized nations with which we are competing. However, the majority of the Nation's small businesses, which so far have not found a way to participate, could make a difference. They make up about 65 percent of the Nation's work force and clearly, reaching this segment of the population is essential.

Specific populations are also at risk to succumb to alcohol related problems. According to the Department's report on the health status of Americans, alcohol is one of the chief causes of illness and death among Black, Hispanic and Native American populations. A recent study has estimated that as many as one-half of our homeless in America suffer from alcohol abuse and alcoholism.

Secretary Bowen's initiative recognizes alcohol abuse and alcoholism as among the top priorities in the Department of Health and Human Services. His advisory panel provided him with a frank, candid report which formed the basis for his 14-point program.

As Chief of Staff, it is my responsibility to make sure the program gets properly implemented. I would like to take a minute and highlight for you what our accomplishments have been to date. There are many ongoing HHS programs which have a natural focus on alcohol abuse and alcoholism. We are currently exploring ways to integrate alcohol abuse and alcoholism into these various programs.

Secretary Bowen has requested from each Assistant Secretary and each operating division head in the Department a summary of his or her ongoing alcohol related activities for the current fiscal year, stressing opportunities for highlighting alcohol related issues and problems in fiscal year 1989. A copy of this survey questionnaire is included in your packet.

The results of this survey will be analyzed and from it we will develop plans to increase attention on alcohol abuse and alcoholism through our existing program mechanisms in target areas which appear to need beefing up.

Research is the cornerstone of our efforts. Dr. Gordis will discuss our research efforts more fully, but I would like to mention a few that are well underway. We are working with the American Medical Association to develop a monograph, "Alcohol and Drug Abuse: A Guide to the Primary Physician."

Also, the Indian Health Service is conducting a symposium and training session on research findings on alcohol abuse for Indian health service providers. There will be 11 regional resident and treatment clinics for Indian youths with alcohol and other drug abuse problems. In the early part of the summer we will have a grant announcement to stimulate research on the assessment of alcohol education and prevention programs. While there are a growing of these programs, we need firm research data on what works and what does not work and the effectiveness of these programs.

The National Institute for Alcohol Abuse and Alcoholism and the National Center for Health Services Research are jointly sponsoring a workshop this September to develop an agenda for research on health economics of prevention and treatment of alcohol related problems. Late this summer a conference will be held on the topic of alcoholism by a number of health care professionals and specialty board organizations. The conference will review content of curricula concerning alcohol abuse, and alcoholism focussing on developing a strategy for the future.

The Assistant Secretary for Public Affairs has major responsibility for carrying out a variety of public information and education activities, including development and implementation of a comprehensive plan to first expand upon existing HHS public affairs campaigns and to incorporate alcohol messages into existing campaigns, to develop linkages with the private sector, and to embark on a series of new efforts, including proposals to develop high risk group targeted campaigns.

Working closely with the National Association of Broadcasters, our public affairs staff is developing a "Housecalls" radio and TV series on alcohol abuse and alcoholism. "Housecalls" is a public service spot that the Secretary does on a weekly basis. The "Housecalls" series on alcoholism will go out to some 10,000 radio stations in the United States and slightly in excess of 1,000 television stations.

Our staff and others will meet in early June with the director and staff of the President's Council on Physical Fitness and Sports to further explore the possibility of developing a series of radio and television public service announcements on the negative impact of drinking on sports activities.

The Office of Substance Abuse Prevention is pursuing with the Drug Enforcement Administration the feasibility of an alcohol and drug abuse prevention program with high school coaches. The Entertainment Industries Council has expressed an interest in working with us on a project featuring alcoholism and alcohol abuse similar to the highly successful Isaiah Thomas anti-drug video of last year. This new project will feature members of the U.S. Olympic swim team.

One of the most exciting of our efforts has been our work with the National Council on Alcoholism to establish a private National Citizens Commission on Alcoholism and Alcohol Abuse, which the Secretary alluded to. The Commission has been established and the members are prominent individuals. They are creative, talented, energetic people from various walks of life and I think the positive impact of this group is limitless. They are going to have their first meeting in August. Although intended to be privately funded in the future, this Commission was initially funded out of non-appropriated HHS funds. The first meeting, as I mentioned, will be in Chicago in August.

In addition to these activities, Dr. Bowen has instructed all operating and staff divisions in HHS to replace the term "substance abuse" with the term "alcohol and other drugs of abuse." Dr. Bowen has sought to bring other Federal agencies and departments into the process. Due to the Office of Personnel Management's efforts, for example, the Federal Employees Health Benefits Program now includes comprehensive alcoholism treatment benefits.

HHS and the Departments of Labor and Commerce are discussing opportunities for disseminating information on employee assistance programs and treatment programs to small businesses and identifying creative ways for small businesses to join in EAP activities. HHS and Education are collaborating in addressing the problem of marketing and promotion of alcoholic beverages on college campuses.

Finally, our second National Conference on Alcohol Abuse and Alcoholism will be held in the fall of this year. The conference will be open to the entire alcohol field as well as the interested public participants. We welcome the participation of Congress and hope that you will join with us in bringing to the American people both the knowledge they need to deal with alcohol related problems and the conviction that together we can make a difference.

Let's work together for the betterment of our future and that of our children. This is a major problem we can effectively address without the necessary infusion of massive new additional resources if only we provide the national resolve and leadership to do it.

Mr. Chairman, you are to be commended for having the foresight to hold these hearings on a subject that touches the lives of virtually every American. I am encouraged that working together great strides can be made in successfully addressing this serious national problem.

At this time, with your permission, we have arranged to show you a sample of our public service announcements that we have developed as part of our public education campaign.

The first is one aimed at 8 to 12 year olds, the "Be Smart, Don't Start" and the second was developed for the Indian Health Service. It is targeted to that group.

The third, never before aired, was done in conjunction with the Country Music Association's 15th anniversary awards. The artists donated their time and services for this effort. And the fourth is a parenting message on alcoholism featuring Bill Cosby.

Chairman GLENN. Fine. Just before they start, just one question. Well, a couple of questions.

How long do these take?

Mr. BURKE. About 10 minutes.

Chairman GLENN. Are we making any progress in getting the networks to put these on any time except between Johnny Carson and the end of Nightline?

Mr. BURKE. We are working on it, Senator.

Chairman GLENN. It does not do much good to have the best spot in the world of Bill Cosby targeted to 8 to 12 year olds and so on if it comes on about 3 o'clock in the morning, and that is where I see these things usually. I hope you are making some progress in that area.

Mr. BURKE. I think we have a commitment. One of the stations is going to devote the entire month of January to pushing this particular initiative of ours. I think it is ABC, but I am hesitant to say that, because I might be wrong.

Chairman GLENN. I hope so.

Secretary **Bowen**. We do have the one for Saturday morning focussed on the children.

Chairman **Glenn**. Good. We can make some progress there.

[Video presentation]

Chairman **Glenn**. Okay. Thank you. Those are very interesting. I hope you can get them on a lot of spots.

Let me say, I apologize to the people in the room here but since we do have TV today on this and they were focused on the TV, I thought it was more important that we go out with that to, I hope, several hundred thousand people all over the country on CNN than turning the TV around here so you could see it in the room here. So I am glad you would bear with us on that. Thank you.

Mr. **Burke**. Senator, I should note that you have—every member has a copy of all of these spots in your packages.

Chairman **Glenn**. Yes, and we will show those. I want to see those. You provided quite a packet of information here, including a video tape for each of us this morning, and I do appreciate that, and all the publications here also, and we will make sure that every one of our Senators on the Committee gets that. Thank you very much for bringing that up with you.

Our next witness is Dr. Enoch Gordis who is Director of the National Institute on Alcohol Abuse and Alcoholism.

Dr. Gordis, welcome and we welcome your testimony.

Dr. **Gordis**. Thank you very much, Chairman Glenn.

My colleagues and I at the National Institute on Alcohol Abuse and Alcoholism are very grateful for the opportunity to present the activities or our Institute. It may seem like a hard act to follow after these very entertaining public service announcements. However, I think it is useful that we saw it in this order, because what these public service announcements are doing is to translate in terms that the American people can understand the results of scientific investigation. And this, of course, is the ultimate purpose of all the science which is conducted at the Institute.

In the few minutes that have been allotted for my testimony, I would like to do the following. I would like to say what the function of our Institute is and how it is organized to accomplish that function, some of the main areas of our science, and finally I would like to conclude with a few short remarks about the relation between the scientific institute and the issues of social policy on one hand, and the treatment community on the other.

To begin, I should point out that the NIAAA is a science institute devoted to research, devoted to the task of finding scientific solutions to the problem of alcohol abuse and alcoholism in our country and to work out the ways to prevent the problems when they start and to ameliorate them once they have been established.

To do so, we have essentially a research function. Most of our budget goes to extramural research; that is, outside researchers across the whole country who apply for research support. A small fraction of our budget goes to the research activities which take place in the intramural program of the National Institute on Alcohol Abuse and Alcoholism on the campus of the National Institutes of Health.

We also have other functions which are important; research dissemination both in print and by computer, as well as curriculum development for physicians and physicians-to-be, and other health professionals and medical schools. In the interest of time I will limit my remarks principally to the research agenda.

In general, our research activities are split into two main categories—the biomedical on one side and the psychosocial on the other, the latter including the very important work we do in research on treatment, treatment efficacy and prevention. The main topics in our biomedical portfolio now are the addictive process, genetics and the toxic effects of alcohol on various organs.

The addictive process research is perhaps the most fundamental because it seeks to answer the main question, perhaps, in all of alcohol abuse, which is, why is it that millions of Americans, decent, intelligent and responsible, do not seem to be able to stop drinking even though they know they are killing themselves? In order to understand that better, we will have to have far greater insights than we have now into the mechanism of addiction.

And among the topics which this slogan or topic includes would be the issue of tolerance to alcohol, the issue of the mechanism by which alcoholics develop withdrawal symptoms, and most importantly, perhaps, the craving for alcohol which is the hallmark of alcohol dependence even among people who have succeeded in stopping drinking. And this craving, of course, is the key precursor for a relapse, which is one of our greatest problems in treatment.

Another area which you are going to hear more about during your hearings, Chairman Glenn, is the area of genetics. We now know from population studies that a large component of the vulnerability to going on to becoming alcoholic once exposed to alcohol is a result of factors yet unknown which are inherited.

The task of research in this discipline for the next 5 and 10 years will be several. First of all, to define large families which have problems so we can see how the disease segregates among different people, to enter into the realm of molecular biology so we can identify which portion of the genome is occupied with this problem of vulnerability, and finally, to learn how the environment and other non-genetic factors intersect with genetic factors to produce the picture of alcoholism in any given individual.

Now, you will be hearing a good deal more about this, I believe, from the program tomorrow so I will simply move over to another area of our biomedical portfolio, and that is toxic effects. It is very important to understand how alcohol damages organs because if we know that we are in a better position, first of all, perhaps to prevent it, and then if not possible to prevent it, to reverse the damage once it has occurred.

Alcohol affects many organs. They were listed very well by one of the speakers in one of the spots. Among the areas we are concerned with, of course, are the damage that alcohol does to the fetus, to the liver, to the brain. Alcohol is a potential carcinogen, and so on.

We have had excellent progress in some of these areas harnessing the new techniques of biochemistry and scanning. And if there is any specific interest on the part of the Committee in these areas I will be very happy to do my best to answer questions.

The other half of our portfolio is not so much in the biomedical area as in the areas of treatment and prevention. To emphasize the fact that the Institute considers this an equally important part of its research agenda, I wish to tell the Committee that, about a year ago, a new division devoted to the topics of treatment and prevention was established at the Institute which is now going at full tilt and we have a large portfolio of projects, both in treatment and as well in prevention.

In the treatment area, the important things have to do with first developing new technologies for an objective marker of drinking so we will be in a better position to have accurate results about the efficacy of treatment. Other treatment issues have to do with important things like the matching of patients to treatment. That is, which components of treatment are most ideally suited to which type of patients? In that way we will be able to make treatments more efficient, more cost effective and more convenient for the patient.

Another area of treatment research which has been especially funded by Congress during the last 2 years has to do with the issue of for whom is inpatient treatment suitable and for whom is outpatient treatment most suitable? And this is a topic upon which we have awarded five grants during the last year and we are hopeful that this will be the beginning of an important and long-term agenda on treatment efficacy.

And finally in the treatment portfolio, there is always the hope that neuroscience and biochemistry are going to deliver us new understanding of the way the addictive process occurs. From that, we should be able to develop potent new pharmacotherapy for alcoholism—and that is something which I think we already have promising results, but I'm looking forward to a good deal more in the future.

Prevention research is perhaps one of the most difficult areas of research in the alcohol field because as everybody knows, the kinds of interventions that one would like to do in prevention do not occur in a vacuum. Rather, they occur in the real world where all sorts of other things continue to happen—TV, the media, school, peer pressure and so on.

To develop research which will prove what interventions are efficacious is one of the tasks of our new prevention branch and we are off to an early and useful start. One of the areas we will be looking into, of course, is the efficacy of school-based programs for which the research base at the present is rather slender.

Prevention research, I should add, really has two main components. One is prevention interventions that have to do with individuals such as school-based programs and so on. The other has to do with the implications for reducing the toll that alcohol takes on our country by various measures of social policy such as the price of beverages, warning labels and various things of that sort.

I want to mention parenthetically, because we do have research in all these areas involving potential social policy, that NIAAA as a science institute provides the science base upon which we hope that those entrusted to making social policy will use in making their decisions.

In conclusion, I would like to say one word about our relation to the treatment community. As a science institute whose function is very much akin to other NIH institutes, for example, such as the

National Cancer Institute or the National Heart, Lung and Blood Institute, and so on, our job is through our research to provide the scientific basis, the early treatment experiments from which the dedicated and skilled practitioners in the outside world can learn and finally utilize these results.

It is important that even the most abstract sounding areas of basic science ultimately have treatment and prevention implications. For example, neuroscience is likely to develop new pharmacotherapy for alcoholism. Our genetics work is likely to provide the basis on which we can intervene in high risk youth before they have gotten into trouble with alcohol and so on.

I am confident that with the contributions of our extraordinary group of researchers both outside and in the Institute that many of the problems you have heard addressed by all the witnesses this morning will be successfully conquered.

Thank you.

Chairman GLENN. Thank you very much.

Mr. Trachtenberg is with us also this morning. He is the Deputy Administrator, Alcohol, Drug Abuse and Mental Health Administration with HHS. I know you did not have a prepared statement. Do you have any remarks you wish to make, Mr. Trachtenberg?

Mr. TRACHTENBERG. No, Mr. Chairman, but I would be pleased to answer any questions that you might have.

Chairman GLENN. Good. Thank you very much.

Secretary Bowen, I believe you have, what? A noon appointment that you have to leave for?

Secretary BOWEN. I have at least a half hour yet.

Chairman GLENN. All right. Fine. Good. That will let us complete our questioning and I appreciate your remaining.

I am concerned about our primary responsibility of providing the funding, providing the wherewithal to run these programs. How much money are we currently spending on alcohol abuse and on drug abuse out of your Department? Do you have that figure handy?

Secretary BOWEN. That is why we brought Mr. Trachtenberg along.

Chairman GLENN. All right. Good.

Mr. TRACHTENBERG. If you take the National Institute on Drug Abuse and consider its budget totally dedicated to drug abuse, their regular budget, not counting their AIDS budget, which is a separate program activity, is $118 million, as requested in the President's fiscal year 1989 budget.

By comparison, NIAAA's total budget is $99 million. There has been a historical difference in NIAAA versus NIDA budgets over the last 15 years since NIAAA and NIDA were established, with NIAAA always funded at a lesser level.

The other two pieces of the alcohol and drug component relate to block grants and the Office of Substance Abuse Prevention. The portion of the block grant that we estimate comes out of the Alcohol, Drug Abuse and Mental Health Services Block Grant for alcohol and drug abuse services is pretty much even, about $125 million per year for alcohol and $125 million for drug abuse. Now, not every state receives it exactly in that way, but overall across the country there is a pretty even split.

In the area of the more recently established block grant authority, the Alcohol and Drug Treatment and Rehabilitation Block Grant, there is it about a 60/40 split; that is about 60 percent of these funds go to provide drug abuse services and 40 percent go to alcohol services. I underscore there the fact that that is a discretionary decision on the part of each State to put the funds where they see the greatest need. There is no limit in the statute in terms of where they should put it. It is their discretion.

Chairman GLENN. Dr. Bowen, what would be an optimum program? What would you like to see? I know we cannot throw piles of money at something like this and make it go away, but surely this is a pittance compared to the problem.

What would be an optimum program? What would be the cost of it as you would see it?

Secretary BOWEN. Well, that is very difficult to put an exact figure on it. Just let me say that I think that the problem of alcoholism and alcohol abuse is probably the sleeper as far as the public health program goes and as far as public awareness of it, so it seems to me that we ought to have more going into the prevention aspect and, obviously, the treatment aspect could have some increased attention.

Chairman GLENN. Do you have any figure you could give us on what you would like to see if you had your "druthers"? What did you request last year when it went to OMB?

Secretary BOWEN. Do you have that figure? He says it was fairly close to what we received.

Senator GLENN. Which is what?

Mr. TRACHTENBERG. The NIAAA request was for $99 million and that is basically what OMB approved for the President's budget.

Chairman GLENN. Dr. Gordis, do you think there is enough money going into this?

Dr. GORDIS. Chairman Glenn, it is always easy for a scientist——

Chairman GLENN. I will throw you a big softball like that.

Dr. GORDIS. Well, I will do my best. Obviously, in an exciting area of science and alcoholism now it is always easy to find new uses for money. I think, given the fact that our budget has increased significantly in the last 6 years—NIAAA has done rather well as far as the percentage increases—and the fact that the field is very active, I think we are making a lot of progress. Of course, the budget reflects the fiscal reality on one hand and the needs of the field on the other.

I could spell out the nature of our research agenda from which people more expert in budgetary matters than I can deduce what it would cost. But on the whole, I think the field has done much better now than it has, say, 5 or 6 years ago.

Chairman GLENN. You are all experts in this field or you would not be here this morning. You deal with it every day. Did you agree with the recent Supreme Court decision that alcoholism is not a disease but is willful misconduct?

Dr. Gordis.

Dr. GORDIS. Well, Chairman Glenn, having read the Supreme Court decision with great interest, I think it is important to point out that even though the disease concept was implicit in much of the discussion, both the majority and the minority opinions carefully stated that they are not ruling on that issue, that they were ruling on a much narrower issue. They were ruling on certain specific things relating to the plaintiffs. For example, whether the plaintiffs had a right to have their cases looked at individually rather than seen as part of a global mix of alcoholics.

The issue was whether these particular plaintiffs were guilty of willful misconduct as against having some sort of a problem for which willful misconduct would be an inappropriate designation. I think it is important that the decision actually did not discuss the issue of alcohol as a disease as such and both sides made a clear point of saying that.

However, there is no question that this topic is somehow implicit in much of the discussion because in the minority opinion, which in my view was brilliantly written, attention was paid to the science of the last 25 years or so, which is continuing to buttress the concept of alcohol as a disease.

In fact, one of the points made in the minority opinion is that the majority had not really looked at the science of the last 10 years which in certain areas—especially in genetics—has been one of the most strong signs of evidence that there is a component of alcoholism which is certainly not willful misconduct and to which people slip into unknowingly, partly because of what their inherited genome is.

So although individual opinions will differ, to me as a scientist and as a clinician, the disease concept of alcoholism is the most plausible explanation of the facts we have at hand.

Chairman GLENN. Dr. Bowen, do you agree with that?

Secretary BOWEN. Yes. Really, the majority's decision was not trying to decide, in my judgment, whether or not alcoholism was a disease. It seemed to me they intimated that it was a disease but it was one over which the victims would have a certain degree of control, and I think that was the essence of their decision.

Chairman GLENN. Dr. Gordis, what kind of progress are we making with regard to medical treatment? And I mean like the use of Antabuse—disulfiram—things like that. Do we have anything, any new promises on the horizon here that we can look to?

Dr. GORDIS. I think we have a lot of promise on the horizon. By and large the improvements in treatment over the last 10 years have been codifications and refinements of things which have been sort of traditional in the field for the last 25 or 30 years.

As you know, Chairman Glenn, alcoholism treatment has arrived at its present status through routes other than the conventional ones which, say, treatment for cancer of heart failure have used. There is a mixture tradition, of the tremendous contribution that AA and its program has made, and the use of certain drugs like disulfiram. The arrival of alcoholism treatment into the medical community is of relatively recent vintage compared to other areas of health.

I would say that we have a lot of promise in the treatment area now. First, the better technology for evaluating our results so we can select which components of treatment are apt for the right kind of a patient. Second of all, various people have developed handbooks, for example, books for the non-pharmacological therapy—such as, relapse prevention technology and life skills training—which have made the delivery of verbal treatment much more efficient and it has been able to be translated from one treatment setting to another with relative ease since this has been described and codified very well.

Both in the national scene and on the international scene we are working on diagnostic schemes so we can have consensus as to the diagnosis of who has an alcoholism problem and who does not. And with this I think we will be in a much better position to judge the results of new treatment research.

Finally, as I said before, I believe that there is already early promising work on new pharmacotherapy which will interfere with the craving for alcohol. Of all the kinds of treatment innovations which I think we can see for the next decade or so, the area of understanding the appetite for alcohol and being able to do something pharmacotherapeutically to intervene will probably be the most potent innovation we can look forward to during the next decade.

Chairman GLENN. Do you have plenty of money to look into that? I know you cannot throw money at it, but in your research do you have enough to adequately fund the promising areas?

Dr. GORDIS. Well, as we said before, one can always use more. And, of course, treatment outcome research and the development of new pharmacotherapy is one of maybe 10 or a dozen terribly important topics within our portfolio. It is very hard to say that we should sacrifice genetics for treatment or sacrifice the cure of liver disease for genetics and so on.

So we have to make a reasonable judgment of how we spread our money into various kinds of topics. Yes, obviously, one can always use more, but I can say that treatment outcome research and the development of new treatment is a major component of what we are doing now.

Mr. BURKE. Senator, we have in our budget a health care improvement fund, the purpose of which is to look at the clinical effectiveness of various treatment modalities, not just in the area of alcoholism but across the whole spectrum. The National Center for Health Services Research has been working with the Alcohol, Drug Abuse and Mental Health Administration and we are going to start looking at the effectiveness of various types of treatment for alcoholism to find out what works and what does not work.

We do know from past demonstrations that the treatment of alcoholism is cost effective and demonstrations that we have done with health maintenance organizations, we found that even after the Federal support of the demonstration was withdrawn, the HMOs continued the program because it was cost effective to the provider.

Chairman GLENN. What is your view of warning label legislation? You mentioned that, Mr. Burke, in your testimony here.

What is your opinion, Dr. Bowen? Would that be effective at all or is that something that we are just nibbling around the edge of the problem?

Secretary BOWEN. Well, as you know, the Anti-Drug Abuse Act of 1986 did include a provision which expressed the sense of the Senate that the Public Health Service should assess a potential educational impact of the health warning labels on alcohol beverage and then report their findings to the Congress.

Well, in response to this provision, the NIAAA did undertake a review of all of the literature on the effects of warning labels on various products and substances which do pose risks in health, and that would include such things as tobacco and aspirin and saccharin and some of the over-the-counter drugs.

So in August of 1987 we did forward that report to Congress and the report concludes, and I quote from it: "Health warning labels do have the potential to affect consumer behavior, particularly in combination with other educational efforts."

Now, you know, the Treasury Department has regulatory jurisdiction in this matter and they have also been requested by the Senate to do the research on this and their report, I believe, is due this fall, in December, so you will be getting the report from the Treasury Department on that.

Chairman GLENN. Dr. Gordis, do you think it is helpful?

Dr. GORDIS. I can only second what the Secretary says. He has very accurately summarized the conclusions of the study which the sense of the Senate directed us to do.

Chairman GLENN. Senator Sasser.

Senator SASSER. Thank you very much, Mr. Chairman.

I want to welcome you, Mr. Secretary, before the Committee this morning and your colleagues here today.

Just let me say, Secretary Bowen, I share your concern about the devastating impact which alcohol brings to many American families, as you have pointed out in your testimony here this morning. There is also an economic impact that comes with alcoholism in the form of lost productivity and cost of recovery. And I have seen in my own office examples of lost productivity on the part of one or two troubled staff members.

Now, in your testimony you estimate that the net impact of alcohol abuse on our economy amounts to more than $117 billion per year. That is a staggering amount. Could you elaborate on some of the elements involved in arriving at this cost estimate and just how you came with this figure?

Secretary BOWEN. These figures were actually from 1983, so I suspect that the cost would be more than the $117 billion that is quoted in my remarks. This would include the cost secondary to loss of productivity, secondary to the health care costs of cirrhosis of the liver and cancer and other diseases that could be associated with it; also, from the amount of crimes that are committed, the amount of accidents on the highway, the amount of injuries that the accidents cause, the amount of homes broken up, the effect on the children and the spouses, and just on and on until you can accumulate the $117 to $130 billion that it is estimated to be the cost.

Mr. BURKE. Senator, that figure was derived from a study that was done for the Department by the Research Triangle in North Carolina around 1984, I believe. We would be happy to submit a copy of the report or a synopsis of the report and where the other figure was derived for the record.[1]

Senator SASSER. Well, thank you, Mr. Burke. I think that would be interesting and helpful to look at that report and just determine the methodology by which this figure was determined. That is a staggering figure, $117 billion. And I have no reason to doubt its authenticity. I was simply struck by the magnitude of it.

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[1] See p. 374.

Mr. Burke, in your testimony you refer to the extent of alcohol abuse among the elderly. Is that a phenomenon that is growing? And is it a phenomena that is growing as a result of a growing elderly population or is it a manifestation of something else?

Mr. BURKE. I think if the incidence were to remain the same, the mere growth in the elderly population is going to cause the problem to increase, since the "babyboomers" are now becoming "elderboomers" and the number over 65 is going to increase considerably and with it the health costs that we are going to have to pick up with Medicare and Medicaid and other Federal entitlement programs.

Senator SASSER. Well, these elderly citizens who are engaging in alcohol abuse now, is this something, a habit that they picked up after they became elderly? Or did they take it into their later years with them, bring it with them from their middle age years?

Mr. BURKE. This I would suspect started before they became elderly, sir. As a person ages, however, the need for medication of other types for other diseases and the effect of alcohol with other medications becomes a problem, as Dr. Windom has alluded to in a recent paper. So it is a problem I am sure does not start when they hit 65.

Chairman GLENN. Jim, will you yield for just a second?

Senator SASSER. Yes, I would be glad to yield.

Chairman GLENN. Dr. Gordis, in your statement didn't you have something about alcohol consumption above 65 goes down?

Dr. GORDIS. Yes. That is true. That is the prevalence of alcohol use among the older population is less than it is among the young. That seems to be a reasonably well established fact. Mr. Burke alluded correctly to the fact that probably the major category of people who are in that category are those whose drinking began as younger people.

But there is a group also who are late starters. It is attributed, perhaps, to becoming widowed or retirement and loss of mobility and the things in which anybody would figure out might be something which might generate the beginning of alcohol use.

There is some contradiction in the data now between the clinical samples and general population surveys on the proportion of late starters to early starters and one of the tasks of research is to understand that better.

I think Mr. Burke was also correct in alluding to one of several problems that alcohol use in the aged causes; that is, there is interaction with many other medications which elderly people are likely to have in large numbers. There is the effect on cognition and memory which, of course, makes the diagnosis of other kinds of problems of that sort more complicated. Alcohol causes depression and depression is a large enough problem in the aged, obviously, even without that. And, of course, with falls and accidents and so on, alcohol is another risk factor.

Because of the immense amount of implications of alcohol on the aging process, both on a biomedical and neuropsychological as well as social sphere, the National Institute on Alcohol Abuse and Alcoholism has just begun funding one of our 11 research centers on alcohol and the aging in which a variety of problems will be addressed, both the epidemiological kind which we have been talking about here as well as some more fundamental ones about the interaction of alcohol in the aging process.

Mr. BURKE. Senator, we do have a documentation that indicates an increasing problem for the elderly in the State of Florida which we will supply for the record.

[The information referred to follows:]

STUDIES ON ALCOHOLISM AND THE ELDERLY

1. Dupree, L.W., Broskowski, H. and Schonfeld, L. (1985) the Gerontology Alcohol Project: A behavioral treatment program for elderly alcohol abusers. *The Gerontologist* 24 (5). 510–516.
2. Galtz, C.M., and Baer, P.E. (1971) Characteristics of elderly patients with alcoholism. *Archieves of General Psychiatry*, 24 (4), 372–378.
3. Dupree, L.W., and Schonfeld L., (1988, in Press) Alcohol abuse among the elderly.
4. Glantz, C. (1981) Predictions of elderly drug abuse. Journal of psychoactive Drugs, 117-126.[1]

Mr. BURKE. If you look at the demographics of the United States just maybe 20 years out, the demography of Florida is basically what the demography of the United States is going to look like. So there is some cause for concern.

Senator SASSER. Let me just ask you this, Dr. Bowen, and the other members of our panel, if they want to chime on this, too.

What is the status of Federal efforts to encourage employers to participate in employee assistance programs to assist the employees in education about the proper use of alcohol, when they are stepping over the line as far as abusing it is concerned, and what the danger signs are if they themselves would be prone to abuse?

Secretary BOWEN. We have joint efforts underway with the Department of Labor to try to reach small businesses with information about the value of employee assistance programs.

We also are working with the other Departments of Government--Treasury, Education and the Commerce and all—to try to get the information and education out to all of the people. I think you have before you a packet of material there that has samples of all of the educational and informational efforts that we are putting forth.

Bob Trachtenberg may have some information on what the ADAMHA is doing that I have perhaps overlooked.

Mr. TRACHTENBERG. Well, NIAAA has been a leader in encouraging the private sector to develop employee assistance programs and, frankly, the private sector has responded quite well in the area of alcoholism and in the general area of mental health.

We continue to have a small research portfolio looking at more effective ways to deliver employee assistance programs and we provide outreach and assistance to the private sector. One of the sectors which has been a little bit slower in starting up employee assistance programs is small businesses and that is a community that we are working with now.

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[1] See p. 340.

Senator SASSER. What are we doing in the Federal service itself to educate employees about the dangers of alcohol abuse? We in the Federal Government are always requiring private industry to do various and sundry things and then I look around and find that we are not doing, Mr. Chairman, what we require them to do.

What are we doing in the Federal service to educate our employees about the dangers of alcohol abuse? And what are we doing about securing treatment for those who do have problems?

Secretary BOWEN. Well, that has been a very recent initiative of our Department. We have been working with the Office of Personnel Management, and working with them we have been able to get the disease of alcoholism included in the insurance treatment program and we are both trying to publicize that to the millions of people on the Federal payroll. Along with that, of course, is all of the education and information material that we put out to the private sector is available to them also and to all of the Secretaries and Agency heads.

Mr. BURKE. Senator, let me just say, sitting next to Dr. Bowen, that is the first question he always asks in the Department: What are we doing here? When we put a report to the President on our long-term care, he said, do we have a long-term care program for Federal employees? He has done the same thing here? We are promoting employee assistance programs. Do we have employee assistance programs with Federal employees' health insurance? At the time we did not. We do now. We do have a long term care benefit for Federal employees.

But he has asked basically the same question you have.

Senator SASSER. Well, Mr. Secretary and gentlemen, my time has expired. Thank you, Mr. Chairman.

Chairman GLENN. Thank you, Senator Sasser.

Senator Roth.

Senator ROTH. Thank you, Mr. Chairman.

Mr. Secretary, what is our goal in this particular program? In the case of drugs, it is to just say no. In the case of smoking I think, again, it is just say no.

Now, we talk about alcohol abuse and alcoholism. What are we seeking to accomplish in this program?

Secretary BOWEN. Well, there is something in it for every age group, I guess. For the children, we have the campaign of "Be Smart, Don't Start," and we are working to publicize that.

Senator ROTH. So there again, it is just say no?

Secretary BOWEN. That is essentially the message. But we have to start down real low because we know that the onset of the use of drugs is down to the 8, 9 10, 11 year age group. So we have got to get the message out early. We also know that those who use alcohol early are the ones that are more apt to go into the use of harder types of drugs.

Then we have to get the message to those who are casual users that enough is enough and that they should know the dangers of continued use. And then for those who are already addicted, the 10 or 11 million people, we are, as Dr. Gordis mentioned, making every effort to find out the best method of treatment and then applying it. But we have got a lot to learn yet in what is the best method of treatment. That research is going on as rapidly as we can get it done.

Senator ROTH. So in a sense you are saying it is very complex because there are different goals for different groups?

Secretary BOWEN. Different goals for each group; right.

Senator ROTH. Now, I have heard it said by some medical experts that, alcohol in moderation is not necessarily bad; is that true health-wise? Would you agree with that?

Dr. GORDIS. That is a very complicated question because one measures the social or relaxing properties of alcohol for which much of the world uses it as against the potential risk of getting into trouble. So I think one cannot make a blanket statement. A lot will depend on the circumstances, how much supervision there is, what is the cognitive state of the person. I do not think anybody is interested in proscribing alcohol for all the elderly.

On the other hand, I think they as well as other segments of the population, as the Secretary has alluded to, deserve to know what the risks are at any stage of their life in getting involved with it. There is no easy summary to your question, I do not think.

Secretary BOWEN. We need to target not only age groups but specific other types of groups in our society. Let me give you a few statistics here that I was not totally aware of until I had reread it. People over the age of 65 do consume a little less than the younger, but then the cumulative effect of it over the years can cause a problem. Women drink less than men and they have fewer drinking problems, but recently for some unknown reason there has been an increase in the amount of drinking between the 35 and 64 year old group. So I think that needs to alert us to find out why and try to prevent.

Hispanic Americans have a higher abuse rate than the general population, and the first generation American Hispanics are drinking more than the foreign born Americans and they have a very high rate of cirrhosis. The American Indian and the Alaskan natives have higher than the general population. In fact, cirrhosis of the liver is the fourth leading cause of death among them whereas it is, I believe, the ninth leading cause for the general population.

The Asian American has a very low level of use of alcohol and in the comparison between whites and blacks. Abstention is more common among blacks than whites and black men who drink are less apt than the whites to drink heavy. But black American women are more apt than white women to drink heavily.

So we have a lot of specifics in there that we may need to try and target to try to prevent.

Senator ROTH. Has there been a change among the younger populations—at least there seemed to be a perception that the youth, the college age, the teenagers were drinking less so-called hard liquor than past generations; is that a fact? Or do you have any figures on that?

Secretary BOWEN. I do not know that statistic. Maybe Dr. Gordis does.

Dr. GORDIS. I think nationally there seems to be some change in that direction. I do not know off the top of my head whether that conclusion applies to the young group in general. In general, as far as the use of alcohol among the young, however, the recent study from Dr. Johnson indicates in the high school survey that there has not been any major change in the use of alcohol among the youth, which is a big problem during the last several years.

Senator ROTH. I would like to go back a moment to a question I raised at the very beginning. A lot of people have wanted to sort of lump together the use of alcohol with the use of drugs. At least there is one difference. One is legal. We may argue whether a substance should or should not be legal but the use of alcohol is legal and the use of drugs, of course, is not.

There has been a lot of discussion in recent weeks about the legalization of drugs. Is there anything to be learned from the problem of alcoholism? As I said earlier, it seems to me maybe you might argue that it would help tangentially as far as crime is concerned, but what about the number of users. Do you see how the legalization of drugs offers any hope based on what has happened in the case of alcohol?

Secretary BOWEN. I personally do not but I do not know that anyone really knows the true answer to that. But I suspect that had the effects of alcohol been known 50 or 60 years ago, maybe it would have not been legalized also.

Senator ROTH. Are you saying that the number of users of alcohol has increased with legalization?

Secretary BOWEN. I do not think there is any question but what the number of users has increased as a result of legalization.

Senator ROTH. Well, I must confess I agree with you. Nobody knows the answer. But that certainly, as one who thinks legalization is the wrong way to go, I think one would run that very real danger.

Secretary BOWEN. Partly, the per capita consumption statistics show has gone down in the last several years, but the——

Senator ROTH. The consumption of what, sir?

Secretary BOWEN. Of alcoholic beverages. It is interesting to note that the highest consumption rate in the country is right here in D.C. with 5.6 gallons, I believe, per individual. The lowest rate is in Utah with about 1.5 gallons, I believe, per individual. So there is quite a variation in the areas of the country.

Senator ROTH. Isn't part of the problem peer pressure and acceptance?

Secretary BOWEN. I think the problem would be, perhaps, threefold: number one, peer pressure; number two, curiosity; and number three, parents do not care.

Senator ROTH. But in the case of alcohol, generally speaking, certainly in Washington, the cocktail party is a very accepted mode of entertainment.

Secretary BOWEN. I suspect that has a lot to do with it.

Senator ROTH. It seems to me that part of the problem that we are dealing with here is particularly the young see it on TV and radio and in magazines and among their friends and parents as an accepted mode of behavior. So in a sense, if you are really going to control, we are talking about changing the mores of this Nation. In a sense we have done that in the case of tobacco, haven't we? Or we are in the process of doing that.

Secretary BOWEN. We recognize the problem of alcohol being shown as a very popular thing to do and we are working with the radio and TV programs to try to get some changes in scripts and so forth so as not to make it look like it is the thing to do to be popular.

Senator ROTH. Well, Mr. Secretary, as I said earlier, I want to congratulate you and your colleagues in the measures you are taking. Much of the problem does seem to lie with education and within the family and that is a tall order. I wish you luck.

Secretary BOWEN. Thank you.

Chairman GLENN. Thank you, Senator Roth.

Let me come back to warning labels again. Do you favor warning label legislation? So we can get that on the record.

Dr. Bowen.

Secretary BOWEN. I personally would favor warning labels but, again, that is out of our jurisdiction. That is in the jurisdiction of the Treasury Department.

Chairman GLENN. Well, what is the Administration policy on this then? Do they favor warning labels or not?

Secretary BOWEN. I think it is silent as far as I know on warning labels. I am just giving my personal opinion that I think warning labels would be effective and I think that it would be well to see them tried. After all, we have warning labels, as I mentioned, on tobacco, aspirin, saccharin, and such things as that.

As many problems that alcohol abuse and alcoholism cause, I think we ought to use every method we possibly can to try to curtail the abuse and use--over use--of alcohol. I am not trying to promote a new prohibition but I do think that we need to warn everybody of the seriousness of the use of alcohol.

Chairman GLENN. I understand that and I know you have to go very shortly. Just a couple of brief questions here.

The White House Conference for a Drug-free America in its draft recommendations has called for, and I quote: "Immediate steps to be taken to ban all alcohol advertising on radio and TV."

Do you support that draft recommendation, Dr. Gordis?

Dr. GORDIS. It is a difficult question to answer from the point of view of science, Chairman Glenn. Obviously, intuitively we have the feeling that there is a mass of alcohol advertising which is addressing in many cases audiences we would prefer not to be drinking at all. Aside from the issue of the tension between the damage that alcohol causes and civil liberties issues, which I am not an expert on, from the science point of view there is a good deal of question yet on the basis of the limited research we have, exactly what role advertising has in accomplishing this.

I do not want to expand—it is late now—but the fact is we do not have conclusive evidence that advertising really initiates drinking among the young. However, the research in this area is extraordinarily difficult and there is a kind of a contradiction between the limited evidence from the science point of view and the tremendous intuitive public idea that advertising must be very potent, otherwise they would not have it.

I do not know the answer to that conflict between science and personal experience yet.

Chairman GLENN. Dr. Bowen, do you favor banning radio and TV advertising?

Secretary BOWEN. I cannot say that I would favor absolute banning but I think it should be controlled and the wording and the types of pictures that they use should be improved. I express concern about the alcohol advertisements in my remarks to the Conference, especially where they depict people in high-risk types of activities consuming alcohol, like swimming, canoeing and these types of things that could be pretty dangerous if one did not have complete control of his coordination.

Chairman GLENN. We are mounting a massive education campaign in this and yet I think there have been some reports and studies, some of which were funded by NIAAA, that indicate that educational campaigns increase knowledge about a problem but they do not usually change a person's habitual behavior.

We are pretty much relying on this type prevention strategy of the increase of knowledge about it. Is there any other approach you are considering? Just knowledge about it does not necessarily change the pattern.

Secretary BOWEN. I think I will ask Dr. Gordis to respond.

Dr. GORDIS. I think you have asked a terribly important question in the whole prevention domain, Chairman Glenn, because as I said, the prevention research area is one of the most difficult. The efficacy of school-based programs, for example, the little research we have here is somewhat in doubt because of the very reasons you said, that knowledge and attitude do not necessarily imply a change in behavior.

One of the tasks will be to develop new interventions and document them with appropriate scientific research. However, in saying that, I think we are faced with the reality that we have a tremendous problem in the country, that there is a need to do something, and so therefore I think the middle ground that we take now in the prevention world, which is exemplified by many of the programs, for example, of OSAP, is the correct one.

We do on one hand what seems to be plausible and make sense and seems to have some general effect and at the same time we continue with the research to tighten up the evidence for efficacy prevention efforts and develop new ones.

Chairman GLENN. Dr. Bowen, I know you have to go. Just one more question.

The President, in his State of the Union message this year, said: "Most important, the number of drug users is down. Our children are showing us that they are willing and able to say no to drugs. We are on our way to a drug-free future."

In April of this year you were reported as having told the President, and I quote again from the press report: "Generally speaking, Mr. President, I prefer optimism to pessimism, as you do, but today I don't know how I can honestly be very optimistic on the war on drugs. It's difficult for me to say we are winning the war on drugs."

Could you comment on the differences between you and the President in this?

Senator BOWEN. I really do not think that there are differences. I sat right beside him when I made it and I made it before the entire Cabinet and I thought that the remarks were reasonably well received. Certainly, there has been some progress. We have had some reduction in the users as far as the survey tells us on the senior high school students. But then when we read in the papers here in D.C. of a murder almost every day with more than half of them due to drugs, and when we see that 10 year olds are carrying guns and committing crimes associated with drugs, and when we see that even though we are interdicting tons and tons of the material, there are still tons and tons coming in, it is my impression that as long as we have a demand for drugs there is going to be a supply.

So I guess I wanted to emphasize my feeling that we had to do more to decrease the demand for drugs if we are going to win the war on it. He is very aware that we are long way from winning the war. I think that he has reason for some optimism but I did not feel that I would be doing service to the President or my country if I did not speak out and state that I thought we were winning a few battles but we had a long way to go yet before we were winning the war.

Chairman **Glenn**. Thank you very much, gentlemen. We appreciate it. The other members of the Committee that could not be here may wish to submit questions. We may have some from staff also. We would appreciate your replying to those in detail and it will be included in the Committee record.

Thank you very much.

Secretary **Bowen**. Thank you, sir.

Mr. **Burke**. Thank you.

Chairman **Glenn**. Our second panel deals with the coordination of alcohol abuse programs.

We have Ms. Anne B. Robertson, Director of the Division of Alcohol and Drug Abuse, Mississippi Department of Mental Health, Jackson, Mississippi, representing the National Association of State Alcohol and Drug Abuse Directors; and Ms. Christine Lubinski, Washington Representative of the National Council on Alcoholism.

Ms. Robertson, if you would lead off please, go right ahead.

TESTIMONY OF ANNE D. ROBERTSON, DIRECTOR, DIVISION OF ALCOHOL AND DRUG ABUSE, MISSISSIPPI DEPARTMENT OF MENTAL HEALTH, JACKSON, MS, REPRESENTING THE NATIONAL ASSOCIATION OF STATE ALCOHOL AND DRUG ABUSE DIRECTORS [1]

Ms. **Robertson**. Thank you, Senator Glenn, and thank you for the invitation to appear before you today to address the role of the states in providing alcohol prevention and treatment services.

I am Anne Robertson and I am the Director of the Division of Alcohol and Drug Abuse in Mississippi. I am appearing before you today as the immediate past President of the National Association of State Alcohol and Drug Abuse Directors, NASADAD.

NASADAD is a not-for-profit organization whose membership is exclusively comprised of the state officials designated by the governors to administer the publicly funded alcohol and drug treatment and prevention system. Before I begin our statement, I wish to express our sincere appreciation and recognition of your interest and leadership in holding this series of hearings to address alcohol abuse and alcoholism prevention, treatment, research and policy issues.

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[1] See p. 139 for Ms. Robertson's prepared statement.

Mr. Chairman, as indicated by your opening remarks, you are already familiar with the range, complexity and seriousness of the problems that result from alcohol abuse and alcoholism. Subsequent witnesses have expanded on the extent and nature of the problem, which were also part of my written testimony so I will not go through those again. But I would like to point out or talk about one alarming statistic. At least it was very devastating to me to hear it.

Several years ago the Surgeon General of the United States issued a report talking about the life expectancies of American citizens, comparing the 1980s to the 1960s. Every age group of Americans enjoys an increase life expectancy, comparing the 1980s to the 1960s, except one, and that is our youth, age 15 to 24, who actually have shorter life expectancies than their counterparts back in the 1960s. This is certainly attributable to the use of alcohol, especially as related to accidents.

The publicly funded alcohol prevention and treatment system relies on financial support from all levels of Government, Federal, State and local. A number of years ago Congress took a leadership role in forging the Federal-State relationship. In 1970, Congress passed the Comprehensive Alcohol Abuse and Alcoholism Prevention, Treatment and Rehabilitation Act, commonly known as the Hughes Act.

In this statute, this law, this legislation, which is a model piece of legislation in my opinion, several important things were created and established and said. Number one, Congress established a public health policy that alcoholism is an illness which is treatable and preventable, not willful misconduct.

Number 2, this law required the governors to designate a state agency as a state alcoholism authority in order to, 3, receive formula grant funds which were made available to each state upon the submission of a state plan, a very comprehensive state plan.

The Federal Government really showed leadership in the enactment of the statute and the things that have subsequently happened as a result of that law. In 1972, Congress followed with the Drug Abuse Prevention Act, a very similar piece of legislation.

Under the Omnibus Reconciliation Act of 1981 which created the Alcohol, Drug Abuse, and Mental Health Block Grant and significantly revised the role of the National Institute on Alcohol Abuse and Alcoholism, and the National Institute on Drug Abuse, the mandate for these state structures was repealed. However, I am pleased to report to you that each state and U.S. Territory has retained the single state agency governmental structure to effectively coordinate state alcohol and drug abuse treatment and prevention services, including the administration of the relevant portions of the ADMS Block Grant.

When the Block Grant was authorized in 1981, the responsibility to the Federal Government for the administration of the Federal Alcohol and Drug Abuse Service's programs would change significantly. The Block Grant authorized by Public Law 97-35 turned complete administration and responsibility for the Federal alcohol and drug abuse service dollars to the states. The transition from categorical to Block Grants for the State alcohol and drug abuse agencies was smooth and did not cause major disruption in services, due in part to a temporary overlap of Federal funding, and also to the experience of the states in already administering the formula grant programs, which are very similar to Block Grant programs.

A major continuing cause for concern and one which has hampered many States' abilities to provide alcohol services to those in need has been the drastic reduction in Federal financial support which accompanied the Block Grant program. Exhibit A, included along with my written statement, provides a comparison of Federal appropriations for alcohol and drug treatment and prevention services from fiscal year 1980 to fiscal year 1988. There was a dramatic reduction.

In an annual survey conducted by NASADAD each State agency director is asked to provide a narrative description of major needs in which resources are not adequate. In the most recent survey on fiscal year 1986 resources, the States responded that major needs regarding alcohol services include the inability to meet the requirements of special populations, including adolescents, women, minorities and the elderly; the lack of adequate detoxification services; and insufficient outpatient programs.

As a sideline here, we have done some research on the elderly and alcohol consumption based on DUI offenders in Mississippi and find a lot of situational incidents of alcohol use among the elderly in relationship to widowhood, retirement and those sorts of things, supporting something that Dr. Gordis said, I believe.

Additionally, NASADAD has taken an active role in promoting the enactment of Federal legislation requiring rotating health warning labels on alcoholic beverages, and we thank you, Senator Glenn, for sponsoring this bill.

I also wonder why we do not have ingredient labeling on alcoholic beverages. I do not think Americans would buy a jar of mayonnaise if they did not know what was in it, but they certainly buy beer and wine and everything else and all they know is the alcohol content by volume.

Although the Anti-Drug Abuse Act of 1986 authorized a new Block Grant for alcohol and drug treatment and rehabilitation services, both the Administration and Congress have encouraged the States to use the majority of these monies for anti-drug abuse activity. Thus, in spite of the additional new Federal funds, the Federal Government has not encouraged expansion of services to meet the needs of those suffering from alcohol abuse and alcoholism.

The States were previously required to report data to the Federal Government as a condition of the receipt of Federal funds, but these requirements were eliminated with the authorization of the Block Grant. States have become increasingly concerned over the lack of data available at the national level, which comprehensively and accurately described the treatment and prevention network and its clients. And while a considerable amount of data was collected by NIAAA prior to the beginning of fiscal year 1982, only limited data has been collected since that time.

As responsible State officials, we believe that a uniform data collection tool and process should be jointly developed and implemented by State and NIAAA representatives with Federal financial support. NASADAD is willing to work with Congress and the Secretary in whatever way we can to assist in enhancing national data collection efforts which will help the alcohol field to better assess the service delivery system and its clients.

In closing, we encourage this Committee to recognize that the publicly funded alcohol and drug abuse services programs which rely on Federal, State and local Government funds, do remain effective providers of low-cost, quality services. We would like to once again express our appreciation for your interest in the alcohol abuse field and encourage the support of your Committee in seeking ways to build the Federal-State partnership.

Thank you.

Chairman GLENN. Thank you, Ms. Robertson.

Ms. Lubinski.

TESTIMONY OF CHRISTINE LUBINSKI, WASHINGTON REPRESENTATIVE, NATIONAL COUNCIL ON ALCOHOLISM, WASHINGTON, DC [1]

Ms. LUBINSKI. Thank you.

My name is Christine Lubinski and I am the Washington Representative for the National Council on Alcoholism. NCA's two leading missions are public education and public advocacy. NCA was founded in 1944, to educate the public about alcoholism as a treatable disease and to advocate for services and public policy in the best interest of alcoholics and their family members. We have 190 State and local affiliate councils.

The history of the Federal alcoholism effort began in earnest with the 1970 enactment of the Hughes Act, a Federal law which created the NIAAA and a network of public treatment services and brought recovery from alcoholism within the reach of thousands of Americans. Without the national leadership framed in the Hughes Act, a characterization of alcoholism as a moral weakness might still dominate the public mind.

In the early days, NIAAA funded treatment services through direct grants to community organizations, the project grant program, and to states through the formula grant program. The first real blow to a decade of progress in Federal alcoholism efforts came with the Omnibus Budget Reconciliation Act of 1981. Treatment services were stripped from NIAAA and moved into the Alcohol, Drug Abuse and Mental Health Services Block Grant administered by ADAMHA.

Treatment funding was reduced by a full 40 percent. We lost valuable information about treatment as Federal data collection efforts came to an abrupt halt. NIAAA was reduced to a small research institute.

Despite this traumatic and abrupt reduction in Federal oversight and support, the public treatment system has survived, if not thrived. But what of the losses? They are immeasurable and include the abandonment of Federal treatment standards, significant gaps in treatment services, the loss of national knowledge about the numbers of individuals seeking public treatment services, their demographic characteristics and the outcome of their treatment.

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[1] See p. 151 for Ms. Lubinski's prepared statement.

NCA's view is that the Federal Government has a responsibility to assess the national scene, identify un-met needs and encourage or mandate the expenditure of Federal dollars to meet the needs of unserved or under-served populations. Now that Federal block grant dollars represent only 20 percent of all public money spent on treatment, national interests can best be served by directing those Federal dollars to provide services for the under-insured and the uninsured as well as those with particular cultural, ethnic or gender needs.

This Federal direction is critical if the treatment needs of all populations—women, ethnic and racial minorities, adolescents, the homeless and the indigent—are to be met. NIAAA slowly increased its research budget as it simultaneously narrowed its focus. In the Anti-Drug Abuse Act of 1986, Congress moved to further reduce NIAAA's role when it removed virtually all prevention activities from the Institute and placed them in the newly created Office for Substance Abuse Prevention.

Despite almost exclusive attention to illicit drugs in the war on drugs, alcohol was included in most of the prevention and treatment components of the bill, if only mentioned obliquely in the debate and the statutory language. Alcohol advocates were somewhat hesitant to argue for further attention since the debate so frequently teetered on the brink of a war on drug users instead of a war on drugs. We were hard pressed to offer alcoholics up as additional victims in the fray.

Moreover, the roughly $600 million in prevention, education and treatment funding was a sufficient sweetener to the $1 billion in military, law enforcement and other drug interdiction efforts to please a constituency desperately in need of those dollars while concerned about the vitriol and hysteria of the overall effort.

Institutional problems frustrate attempts to forge a comprehensive national policy to reduce alcoholism and related problems. The history of Federal involvement in alcohol is a history of fragmentation, non-communication, and agencies working at cross purposes.

Although NIAAA represents the heart of the Federal alcohol effort, NIAAA lacks any authority over regulations or Federal policy of any kind governing beverage alcohol. Despite NIAAA's placement within the public health service, there has been complete Government resistance to utilizing the public health model to address alcoholism and alcohol-related problems.

And what is the legacy of this limited view? During debate of the Hughes Act in 1969, estimates of the cost of alcoholism and related problems to the Nation were pegged at $12 billion. Today the cost of these problems is slated to be closer to $120 billion a year.

And how have Federal institutions responded? While NIAAA funded treatment programs in the past and today funds research to identify a genetic predisposition to alcoholism, the Bureau of Alcohol, Tobacco and Firearms continues to promulgate regulations in the economic interest of the alcohol beverage industry with little regard for the impact of any policy on the public health.

The Food and Drug Administration diligently monitors the dissemination of legal drugs to physicians and consumers while the Nation's favorite legal drug—alcohol—continues to be exempt from any of the consumer information or protection initiatives implemented over the last two decades.

The Federal Trade Commission has repeatedly refused to investigate or further regulate alcohol advertising. Institutional problems call for institutional remedies. NIAAA's 15 years of experience should inform all Federal alcohol policy. Congress should delegate increased authority over alcohol regulation to the Department of Health and Human Services, either through the already existing regulatory mechanism of the Food and Drug Administration, or through the creation of a new regulatory mechanism within NIAAA.

I would like now to summarize the remainder of my testimony by offering NCA recommendations for a comprehensive national plan to address alcoholism and related problems. In the area of NIAAA initiatives, increase funding for data collection, prevention and psychosocial research and the establishment of a long overdue national research center on women, alcohol and other drugs; the establishment of an inter-governmental mechanism allowing NIAAA to communicate research findings and policy recommendations to other Federal agencies and directly to Congress; the re-establishment of a strong clinical training program to educate physicians, counselors, and other clinicians about the latest research pertinent to clinical practice.

In the area of prevention initiatives, immediate Congressional action to mandate health and safety warning labels on all alcoholic beverages as contained in S. 2047; enactment of a law requiring equivalent time for health and safety messages on alcohol when alcohol ads are aired on radio or TV, and that is the only way those public service announcements, Senator Glenn, will get on prime time, I assure you; significant increases in alcohol excise taxes and increases in tax rates across the beverage classes as a vehicle to reduce alcohol consumption and institutionalize a tax policy which taxes alcohol as alcohol, regardless of the beverage type; a transfer of regulatory authority over alcohol from BATF and FTC to FDA, or the establishment of a regulatory authority within NIAAA to ensure that public health considerations are an integral component of Federal alcohol policy; increased funding and jurisdictional authority for the Office for Substance Abuse Prevention to extend prevention initiatives to all at-risk populations.

In the area of treatment, substantial increases in funding for the Block Grants with targeted funding for indigent and under-served populations, including the homeless, ethnic and racial minorities, adolescents and women. Increasingly, publicly funded programs are forced to compete with the private sector for paying clients to keep their doors open. This reality generates unnecessary competition with the private sector and reduces treatment opportunities for the under-insured and the uninsured and Federal dollars should be used to serve those people.

The enactment of a Federal law requiring the establishment of employee assistance programs for members of Congress and their staffs in the House and the Senate. No such programs currently exist although the rest of the Federal Government is required to have them.

In the area of Federal reimbursement policy for the provision of alcoholism and drug treatment, a mandatory alcoholism and drug treatment benefit for all health insurance plans offering benefits to Federal employees. Although it is true that the Office of Personnel Management has urged all Federal plans to offer those benefits, it is not required that those benefits be mandated and there are very few Federal workers, or any other kind of worker, that look at a health insurance plan and say, in case I become alcoholic, perhaps I ought to carry a little coverage for that. So the only kind of alcoholism benefit that works is one that is included as an integral part of the plan; expansion of Federal Medicare and Medicaid coverage for alcoholism and drug treatment to include reimbursement for services and free-standing residential facilities and enhanced outpatient coverage.

And finally and perhaps most important to the field at large in the reimbursement area, an amendment to S. 1265, Senator Kennedy's Health Insurance for All Workers Act, to include statutory language requiring the inclusion of comprehensive inpatient and outpatient benefits for alcoholism and other drug treatment.

In the area of civil rights protections for alcoholics and other drug-dependent people, we need an amendment to a law governing the Veterans Administration allowing for an extension of educational benefits to alcoholic and drug-dependent veterans on the grounds that alcoholism is a disease and an eligible disability. We need the adoption of a sense of Congress resolution placing Congress on record unequivocally in support of alcoholism as a disease entity. And we need the adoption of anti-discrimination protections to cover disabled persons, including alcoholic and other drug-dependent people under the Fair Housing Act.

If there is a single common denominator to all of these issues it is the crying need for Federal leadership, especially Congressional leadership. A former NCA official characterized the current state of affairs by saying that the Federal Government had taken the first step in Alcoholics Anonymous; they had admitted they were powerless over alcohol. I think we may need to move on from there.

In 1984 we lost the Senate Alcoholism and Drug Abuse Subcommittee, and this Subcommittee was combined with the Subcommittee which addresses children and family issues. In the last 2 years we have had only one Senate hearing on our issues, a Subcommittee hearing on children of alcoholics and drug abusers.

In the current reauthorization cycle there have been no hearings in the Senate on NIAAA, NIDA, or the Block Grant, although sweeping changes are proposed in both the Senate and House legislation. This series of hearings, under the leadership of this Committee, are the single most concerted attempt to review alcohol policy that our constituency has seen in the last 8 years. Comprehensive anti-drug legislation is again on Congress' agenda, presenting new opportunities to ensure the inclusion of policy and funding measures to address alcoholism and alcohol related problems.

This Committee has an important opportunity to refocus the Nation's agenda toward a comprehensive and humane plan to reduce alcoholism and related problems. The National Council on Alcoholism stands ready to work in concert with you to meet this challenge.

Thank you very much, Mr. Chairman.

Chairman GLENN. Thank you very much, Ms. Lubinski.

Do you have sort of an ideal alcohol program as you see it? What you would like to see done at the Federal level? Do you have that outline?

Ms. LUBINSKI. I think what we would view as the ideal alcohol plan is contained in the recommendations I just offered. But I guess if I could single out any one thing, it would be for increased coordination and a change in jurisdiction so that policy governing the availability and labeling and advertising of beverage alcohol was in an agency that had some concern for the public health.

Chairman GLENN. Under the Block Grant program, it meant a loss of national data on the numbers of persons seeking public treatment services, their demographic characteristics, outcome of their treatment. In your experience, have the States on their own tried to develop data in these areas? What have we lost under data collection?

Ms. ROBERTSON. The States participate in a State alcohol and drug abuse profile information system that is conducted by NASADAD. That is basically all the data of a national level that is available, to the best of my knowledge. NIAAA and NIDA both stopped both of their data collection processes. The States, in the ensuing 8 years, although our data collection systems are similar enough to participate in that national survey that I mentioned to you, nonetheless there has been some drifting and there are some dissimilarities. It is not easy to compare Mississippi with Missouri or any place else any more our data systems are so different.

Chairman GLENN. Well, there is no requirement to collect data now as a condition of receipt of Federal funds; is that correct?

Ms. ROBERTSON. That is correct, yes, sir.

Chairman GLENN. Well then, is the collection of data on this very important area left just to the whim of the States? If they want to collect data and submit it, they can? If they do not want to, they do not? Is that the way it runs?

Ms. ROBERTSON. Basically, sir, yes. I think the Block Grant puts very few requirements on the States, very few at all, which I think makes, in my opinion, these funds very vulnerable because there is not a data base substantiating what is going on and what is happening with these funds.

Chairman GLENN. Does the Federal Government provide any guidelines to the States on the use of Block Grant funds?

Ms. ROBERTSON. Very few. We submit a plan, a small plan for the utilization of the funds. The basic guidelines are in the language of the Omnibus Reconciliation Act in the law itself and the guidelines do not go beyond that. In other words, I do not know what public policy Congress is expecting to be implemented with the Block Grant funds. I do not think that is apparent to those of us who are trying to do that sort of thing at the State level.

Chairman GLENN. Well, do the States then just make up their own criteria when they are deciding how to spend resources from the Block Grant for alcohol-related servicing programs? Do they just decide what they want to do and that is it?

Ms. **Robertson**. Only within the limits of the law which requires that the money be spent for alcohol and drug abuse activities, 35 percent for alcohol, 35 percent for drug, and 20 percent for prevention. And then there are some assurances——

Chairman **Glenn**. What is the percentage again?

Ms. **Robertson**. Thirty-five percent for alcohol activities, 35 percent for drug activities, and 20 percent for prevention. That, of course, adds up to 90 percent. What happens with the other 10 percent is not addressed in the law.

The States have continued the use of this money in much the way that they did the alcohol and drug formula grant. I have to go back to the 1970 Hughes Act, which was in place for 8 or 9 years before the Omnibus Reconciliation Act. I think some of the residuals of that Act at the state level are what has kept the thing together for the last 8 or 9 years, kept the State network together.

Chairman **Glenn**. How have the States dealt with the reduction in Federal financial support which accompanied the ADMS Alcohol, Drug Abuse and Mental Health Services Block Grant program? The funds were reduced.

Ms. **Robertson**. How have they dealt——

Chairman **Glenn**. Did they just reduce services?

Ms. **Robertson**. In some instances there has been a reduction of services. There was an overlap of money, which helped us through the first year or two in transition in that some monies were coming down through formula grant and categorical project grants across the country before the availability of the Block Grant. That eased the transition in some ways.

I think it is like crying babies are being attended to, in a way. The money is cut, you know, and a lot of the activities in the area of prevention, things that States would like to have done, are not getting done right now. And too, State legislatures in some instances have been able to come across with appropriations to replace some of the lost funds, but that is not totally so across the country.

Chairman **Glenn**. Well, you and Ms. Lubinski both addressed this, but I will ask you the question first and then turn to her. There has been much concern recently about the de-funding of public programs, in particular the public programs in nonprofit organizations have been actively recruiting clients who can pay for services as opposed to those people who lack insurance altogether or whose insurance does not cover treatment services.

In sum, it seems that we may slowly ignore altogether the indigent population in need of alcohol treatment services. Have you noted this trend? If so, what are your recommendations to stop this pattern? What are you doing about it? Anything?

Ms. **Robertson**. Senator Glenn, I am most familiar with that situation as it impacts on Mississippi. At the NASADAD level we have not particularly discussed that, to my knowledge. If I understand what you are asking me, what happened, the private sector——

Chairman **Glenn**. Well, basically, as Federal funds have gone down, you have been left with your own organizations or nonprofit organizations or others that try and deal in this area. As there are less Federal funds, then you have to keep your organizations going and you have to keep the institutions going, the homes or whatever you are putting people into for treatment. If there are not funds there, you have to go for people that can pay for that service and yet there is a great number of people out there who cannot afford that kind of service that we were dealing with when we had better Federal funding.

Ms. ROBERTSON. Yes.

Chairman GLENN. Now, how are you dealing with that.

Ms. ROBERTSON. In the publicly funded area, which would not be the same as the other nonprofit organizations, we continue in my state to serve the indigent first. What happens is in the for-profit agencies quite often after insurance coverage is utilized, whatever is used up, so to speak, by an individual then, unfortunately, they turn those people over to us in the public sector.

I do not know if I am—Christine, maybe you can answer it. I do not know if I am getting at what you are asking me, Senator Glenn.

Chairman GLENN. Ms. Lubinski, would you like to comment?

Ms. LUBINSKI. Well, the reality of what happens in a lot of programs is they receive funding from a number of sources; some Federal money but that comes down through the States. Frequently these programs do not know they are Federally funded. State money, county money, sometimes private contributions, but it is not enough to meet the bottom line so they are forced in addition to taking the people they were set up to serve, which would be, presumably, indigent people or under-insured people, to recruit paying clients to make up that bottom line.

That situation has also been exacerbated by an increasing number of DWI convicted people being mandated to attend these public treatment programs as well, so that there is less room for the self-referred or the doctor-referred public client in those programs.

It seems to me we need to have programs that are set up and fully financed exclusively to serve under-insured and uninsured people and that given the shrinking percentage of Federal money that goes into the whole pot, we need to look at that percentage and say, how can those Federal dollars best be served? And I would argue they can best be served by looking for gaps in the system, namely services for the indigent and some other special populations and saying that is where that money should be spent.

Senator GLENN. What do you see as NIAAA's role? You were quite critical in your statement of NIAAA's functions being changed around somewhat. On page 3 of your testimony, you said, the first real blow to NIAAA as the focal point of the Federal alcoholism effort came with the Omnibus Budget Reconciliation Act of 1981. Another sentence there, treatment funding was reduced 40 percent. The demise of the direct Federal support for services came 5 years too early to ensure stability of the system.

You go on, on the following page, to list some of the losses that occurred, loss of data and other things there. The new federalism hit NIAAA just as Federal treatment standards were being developed. None now exist.

As I understood from what Dr. Gordis said a little while ago, his mandate at NIAAA now is mainly the scientific and the research area. Now, was his organization seriously diminished, from your viewpoint, from what you state here? And is someone else picking up what his prevention and treatment program was previously? Or do I misunderstand that? Was his mission changed?

Ms. LUBINSKI. His mission was greatly diminished and now is not only primarily research but from our perspective, and many other organizations would agree, inordinately biomedical focused so that the kind of research and guidance that is coming out of the Institute does not have a very immediate function for professionals in the field, whether you are in the clinical level or a prevention advocate.

I mean, the neuroscience stuff is extremely important but for those people who are in the business of treating alcoholics today, who are in the business of trying to do prevention work today, the NIAAA offers very few resources for that community. I mean, they used to promulgate standards for employee assistance programs for counselors.

Chairman GLENN. Well, who picked that up? When they lost that, who was supposed to pick up that mission that they had been performing that they no longer had? No one?

Ms. LUBINSKI. No one.

Chairman GLENN. Was that left up to the States, then, with the Block Grant programs to develop that?

Ms. LUBINSKI. Presumably, although, here States were handed 40 percent less money and said to carry on this treatment system and in addition provide all these other services for your constituency.

Chairman GLENN. In your testimony, you say the Anti-Drug Abuse Act of 1986, "breathed new life into the Block Grant program."

How has the Act affected both alcohol and drug treatment services and what has been the primary focus of the Act since 1986?

Ms. LUBINSKI. I think in the area of treatment I also say in my written statement that prior to the Anti-Drug Abuse Act we had flat level appropriations for 5 years. We just could not get Congress on either side of the aisle and either House to be interested in the Block Grant. It is my view, it is because many people saw it as a Federal revenue sharing program. They did not have any control over the money. They did not know what was done with it so who cared?

The most important thing, I think, the Anti-Drug Abuse Act did was direct a lot of badly needed money into treatment. While we were talking about trying to reduce drug abuse and alcoholism in this country there were waiting lines in many parts of the country to get into treatment. We could not even provide treatment to those people who were asking for it.

And I think that it is important to note that in many places in the country and many treatment programs, alcoholic and other drug-dependent people are treated together, so that despite what we felt was an inordinate emphasis on drugs, when it comes to treatment, frankly, there are very few pure alcoholics or drug addicts these days and they are very frequently treated in the same treatment setting. So that what benefits drug-dependent people also benefits alcoholics.

Chairman GLENN. Well, do you have any suggestions on what, if you were sitting in our position here, what would be the first, second, third steps you would want to take to improve our approach to the whole alcoholism problem?

Ms. LUBINSKI. Well, I would direct a whole lot more money to people and a whole lot less to hardware, for one thing. It seems to me the money that is spent on interdiction is having very little return and alcohol and drug problems are growing every day in our community.

I would do what I could to reduce the distinctions between alcohol and other drugs. To suggest that it is only these other drugs that are in somebody else's neighborhood that are a problem, and look here at home at what is going on with alcohol and how alcohol in fact contributes to other drug use.

Chairman GLENN. Well, that is one of the things we are trying to do with these hearings and one of the reasons that we set them up was because with all the emphasis on the hard drugs and the illegal drugs, as good as that is, and I am all for all the programs of cooperating with foreign countries to try and cut down the drug production, try and interdict it once it is out of their borders, try and intercept it before it gets in our own borders, try and intercept it before it gets to the communities, and do the education programs and all the things that we are trying to do.

That, of course, is the big item right now. It is the big issue of the year and yet, if you look at the figures on the numbers of people and families and homes being affected by alcoholism, the legal drug, why, I will not say it dwarfs the other problem, but it makes the other problem look a little small by comparison, if our figures are even half way right. Even if they are off by 50 percent, it is still a much greater problem with the legal drug of alcohol and we want to try and deal with it in some way here.

I do not know whether these programs that were outlined by the previous panel are adequate or not. We may want to get back to you with some suggestions or questions from our other Senators or staff members and address this is some more detail. We cannot possibly cover everything in just 2 or 3 hours, 2½ or 3 hours at a hearing here with a couple of panels. So we may want to get back to you and call on your expertise for more guidance in the future.

We thank you very much for being here this morning.

Ms. ROBERTSON. Thank you very much, Senator.

Ms. LUBINSKI. Thank you.

Chairman GLENN. The hearing will stand in adjournment subject to the call of the Chairman.

[Whereupon, at 1:05 p.m., the Committee adjourned subject to the call of the Chair.]

ALCOHOLISM: BIOLOGICAL AND ENVIRONMENTAL DETERMINANTS

THURSDAY, MAY 26, 1988

U.S. Senate,
Committee on Governmental Affairs,
Washington, DC.

The Committee met, pursuant to notice, at 9:40 a.m., in room SD-342, Dirksen Senate Office Building, Hon. John Glenn, Chairman of the Committee, presiding.

Present: Senators Glenn and Sasser.

OPENING STATEMENT OF CHAIRMAN GLENN

Chairman Glenn. The hearings will be in order.

Good morning and welcome to the second of the four hearings that the Governmental Affairs Committee will be holding on alcoholism. At yesterday's hearing this Committee heard testimony from the Administration, a representative from the State Alcohol and Drug Abuse Directors, and a representative of an alcohol abuse policy organization on what the Federal and State Governments are doing in the areas of alcohol abuse research, treatment and prevention.

It is clear from the testimony we heard yesterday that while the Administration's position on alcoholism research and preventi are very admirable, there are certain features that are troubling. For example, the Administration admits to sending mixed signals on the subject of alcohol warning labels, as one example.

We heard from Secretary Bowen that while the Administration does not as yet have an official position with respect to alcohol warning labels, he personally was in favor of them.

We would appreciate knowing what the Administration's position is on the alcohol warning issue since a bill has been introduced in the Senate to require warning labels on all alcoholic beverage products sold in the United States.

We also received testimony from the National Council on Alcoholism and the National Association of State Alcohol and Drug Abuse Directors that contained numerous suggestions about ways to improve the Federal/State partnership in alcoholism treatment and prevention. The Governmental Affairs Committee staff will be reviewing their suggestions carefully and will make recommendations to me regarding these proposals.

I think while we talk about some of the programs in general terms, we need to remember the tragedy that is occurring in our country because of alcoholism. We have so much attention being paid to the hard drug problem, the illegal drug problem—and I agree with the attention being given to that—but if you talk about numbers of people and families and the tragedies of homes and broken homes and lives wrecked, we know that there are some 100,000 to 200,000 alcohol related deaths every year in this country.

We talk about maybe 4,000 to 8,000 hard drug deaths. That indicates the difference in the enormity of the problem. Automobiles out on the road, 40,000 to 50,000 auto deaths per year, half of those are alcohol related. We thought it was a complete tragedy in Vietnam, and it was—57,000 people killed during the 10 years of the Vietnam war—and yet we have alcohol related deaths, a minimum of 100,000 a year, almost double what we did in the whole 10 years of Vietnam.

One out of four families is affected by someone in the family circle that is involved with alcoholism. Some 10 million Americans that are alcoholics and another 8 million that are estimated to be serious problem drinkers. So 18 million Americans are affected by this. So I think while we talk about our statistics we need to remember the tragedy that is occurring in our midst every day just from this one issue, and that is the reason that this Committee wanted to address the problem.

The focus of today's hearing is on the contribution of inherited and environmental factors to alcohol abuse and alcoholism. As I stated at yesterday's hearing, alcohol abuse and alcoholism are among this Nation's very most serious public health problems. I just gave some of the examples of that which I will not repeat again.

Clearly, in order to reduce the cost of alcohol abuse to society we must find ways to design intervention, and more than that, prevention programs that can identify persons at risk of becoming alcohol dependent before they are exposed to alcohol. We must also apply the results of biomedical and psychosocial research into innovative treatment programs.

We are truly privileged to have with us today the leading investigators in the areas of biomedical and psychosocial research on the causes of alcoholism. We hope to learn from our witnesses the latest research findings on the inheritance of alcohol dependent tendencies in male and female children of alcoholic parents, how the environmental or social context may contribute to problem drinking, and screening techniques that could be used in the future to identify children at risk of becoming problem drinkers before they have been exposed to alcohol.

We would also like to learn from our witnesses how the results of their research could be used to design better alcohol abuse treatment, intervention, and prevention programs. So I thank you all for coming today and look forward to a great day of testimony here. We appreciate your being here.

We have three different panels this morning. The first panel will take up the biological basis of alcohol dependency; the second panel on behavioral and environmental determinants; and panel 3, the biological markers for alcohol dependency and intolerance.

Our first panel will take up the two aspects of this; one, the sons of alcoholics, second, the daughters of alcoholics. And first we would welcome Dr. Donald Goodwin, Chairman, Department of Psychiatry, University of Kansas School of Medicine at Lawrence, Kansas; and second, on the same panel, Dr. Barbara Lex, McLean Hospital, Belmont, Massachusetts.

Dr. Goodwin, we welcome you this morning, welcome both of you this morning, and look forward to your testimony. As I have to tell people at these hearings, please pull those mikes up very close. They are not the very best, and if you will pull those up tight everyone will be able to hear you.

So, we welcome you to the hearing this morning and look forward to your testimony, either in an abridged version or in its entirety. In either event, the entire statement will be included in the record.

Go ahead, sir.

TESTIMONY OF DONALD W. GOODWIN, M.D., CHAIRMAN, DEPARTMENT OF PSYCHIATRY, UNIVERSITY OF KANSAS SCHOOL OF MEDICINE, LAWRENCE, KS [1]

Dr. GOODWIN. Thank you, Senator Glenn.

My job this morning was to briefly review the evidence that alcoholism in men may be influenced by heredity. The first piece of evidence that bears on this comes from family studies, many of them through this century, which indicate—all of them, without exception—that children of alcoholics have a rate of alcoholism 4 or 5 times higher than children of nonalcoholics. Translated into figures, perhaps 20 to 30 percent of the male offspring of alcoholics develop alcohol problems sufficiently severe to be alcoholic, and about 5 percent of the daughters of alcoholics.

So alcoholism runs in families. This has been known for centuries. In fact, it is one of the few—possibly the only—aspect of the subject of alcoholism about which one can achieve almost total agreement. It definitely runs in families. Having alcoholism in the family constitutes the highest risk factor for alcoholism that is known. And with regard to prevention, I think knowledge of this should be widely circulated.

Now, not everything that runs in families is inherited. Speaking French runs in families; it is not inherited. Voting Republican runs in families; it is not inherited, probably. Blue eyes run in families and is inherited.

So the question about something that runs in families is how much is due to inheritance and how much is due to learning, to either or both or neither. It is what is called the "nature/nurture" problem. So we start with the well documented and totally accepted observation that alcoholism runs in families. And then, beginning in the 1970's, people became interested in trying to identify hereditary factors.

The approach generally used involved the opportunity to study adoptees. In the case of adoptees you have the opportunity to study individuals who are separated from their biological parents, raised by adoptive parents, and if they are separated early in life they have little contact with their biological parents.

In the case of alcohol studies, this consists of studying the children of alcoholics who were separated from their biological parents in infancy, raised by unrelated, moderate drinking or nonalcoholic adoptive parents and had no environmental exposure to alcoholism, in the family, at any rate. And so the question is, will the transmission of alcoholism continue to exist in these children who had no contact with their biological parents? Will it continue to exist despite this lack of contact?

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[1] See p. 171 for Dr. Goodwin's prepared statement.

Now, there were three adoption studies of alcoholism launched in the 1970's in three different countries; one in Denmark, one in Sweden and one in Iowa, which is a State in the United States. It is very hard to do adoption studies in this country, I might add, and this is the reason that two of the three studies were conducted abroad. The Danish study was funded by the United States Government.

In the case of the three studies, they came to essentially the same conclusion. This was that alcoholism continued to run in families, even when the children were separated from the biological parents and raised by adoptive parents. It did not seem to make any difference. The rate was just about the same, namely, about 4 or 5 times greater than in the general population or in control groups of adoptees.

This was the general finding of all three studies based in three different countries using three different approaches and samples and so forth, and there was a remarkable consistency across these studies which were published more or less in the late 1970's.

In our case, I was involved in the Danish adoption studies, and we studied the brothers and sisters of the children of alcoholics, some of whom had been adopted and some of whom had been raised at home by the alcoholic parents with all the disruptiveness and trauma and opportunity to emulate your drinking parent. Some of them were raised at home and some of them were adopted and raised by non- or moderate drinking adoptive parents.

We found no difference. It did not matter whether you were raised——

Chairman GLENN. Found what? No difference?

Dr. GOODWIN. No difference. It did not matter whether you were raised at home, with regard to the incidence of alcoholism.

It still was about a factor of 4-fold, 5-fold greater than in the general population.

Chairman GLENN. If you had had alcoholic parents?

Dr. GOODWIN. Yes, if you had alcoholic parents it did not matter whether you were raised by the alcoholic parents or whether you were raised by nonalcoholic adoptive parents. You could conclude, I think, simplistically conclude—because I am sure it is not true—that it does not matter much who you are raised by if you have alcoholism in the family, and particularly if you have a lot of alcoholism in the family.

Chairman GLENN. Did that apply also when—I know I should let you finish your statement, but I am interested in this point—did that apply even when the parents, the adoptive parents, were teetotalers, no alcohol at all

Dr. GOODWIN. Yes.

Chairman GLENN. They still had the same tendency?

Dr. GOODWIN. The characteristics of the adoptive parents did not seem to have any bearing on whether their adoptive children drank or how much they drank. There seemed to be no relationship. There has been a lot of emphasis on—and today you will have people here this morning to talk about environmental factors. I just want to quote this study, which was also essentially the findings of the other two adoptive studies, that the environment did not make much difference if there was a strong hereditary factor.

If there is a weak hereditary factor--I think, to bring the studies up to date suddenly, going to the end of my remarks immediately—if there is a weak hereditary factor or no hereditary factor, then environmental circumstances such as poverty or psychological problems or stress may be related to alcoholism, but if there is a strong hereditary factor, the environment does not seem to make much difference, just to round off and summarize the studies that have been done so far.

Chairman GLENN. How about the other way? How about adopted children of nonalcoholic parentage who then were in an alcoholic adult home?

Dr. GOODWIN. This happens so rarely. They do not let people adopt children who are drinkers, as a rule, and you cannot find a sample large enough. It has been approached, and you can do it in animals, by the way, but in people certainly in the three countries where we did the studies, we attempted to do such studies where we would study families where the adoptive parents did the drinking but the children, as far as we knew, came from non-drinking parents, but we could not accumulate enough people in the sample to reach any conclusions. They screen people who adopt children pretty carefully in Denmark, certainly.

The adoption findings all came out about the same time in the late 1970's and the fact that they were so consistent I think generated a lot of interest in this line of research. I do not know what proportion of the NIAAA budget is now dedicated to studies bearing on genetics and alcoholism, but a fairly large part, I suspect.

Generally, the research that has been encouraged from these findings of the 1970's, the adoption findings, has been in two directions. One is to study the children of alcoholics before they start drinking. If you are comparing alcoholics with nonalcoholics, one of the problems is that alcoholics drink more than nonalcoholics. That sounds facetious, but this, as a matter of fact, poses a big problem because then you do not know whether you are studying the consequences of the drinking or something that came before the drinking.

So by studying children of alcoholics—particularly the male children, because they are at higher risk than the female children of becoming alcoholic—you study them before they start drinking and then you wait 20 years and see which of them develops a drinking problem, with the expectation that perhaps 20 to 25 percent of them will. And then you look back at the data you accumulated before they started drinking and you look for correlations in the hope of finding predictors of future alcoholism.

These studies are in the first phase now. There are perhaps a dozen of them being supported in various countries, primarily in this country, so-called high risk studies, meaning that the sons of alcoholics are at high risk for alcoholism and therefore you study them now before they start to drink and then you keep studying them until they either do not drink or do drink or somewhere in between, and then you look for predictors.

As I said, the only strong predictor we have now of alcoholism is a family history of alcoholism. We need better predictors, because when we have better predictors, not only may we improve our ability to head off the problem before it gets underway, but also better predictors means better ideas as to causation. And we are really not going to have anything too successful, I suspect, for this problem until we know more about causation.

The fact is that most people in this country drink. About 1 out of 10 or 1 out of 12 get into trouble from it and we do not know why the 1 out of 10 or 1 out of 12 get into trouble, except that the family history is a contributory factor.

So these so-called high risk studies have gone through the first stage, and I would like to make a point here, and that is that these are longitudinal studies and the history of medicine and social sciences is a sad history with regard to longitudinal studies. They are often launched and rarely completed. The investigators die, lose interest, run for political office or—I do not know what happens to them—but various——

Chairman GLENN. Wish a better fate than that on them. [Laughter.]

Dr. GOODWIN. Or, you know, marry somebody who is not interested in this. Various kinds of things can happen so that the original data, this very expensive body of data is collected and gathers dust in a basement some place and is forgotten about because somebody like Dr. Vaillant, who will be talking later, just happens to find this data and then goes out and finds the people.

But unless a longitudinal study is longitudinal, and that means unless there is a long term follow-up, as in the case of the Framingham study where every year they have to fight for funds——

Chairman GLENN. Are the studies you are talking about now equally applicable to men or women or male/female, children?

Dr. GOODWIN. Absolutely. Any study designed to be a longitudinal study should be designed to be a longitudinal study. That is, the payoff is the follow-up and irrespective of the subject. I am now talking about alcoholism studies, since that is what we are talking about. But the payoff is the follow-up and the follow-up is 10, 15, 20 years down the road for the more recently started studies.

As I said, there are about a dozen such studies in existence of cohorts of groups of people, children of alcoholics, some of them as young as 9 or 10, most of them in their late teens, and you now need to study them in their 30s and 40s. One of the problems is in getting the funding agencies and the peer review system to realize that there has been a huge investment made already in this and that—somehow there should be a mechanism that longitudinal studies will be supported over a 20-year period, realizing that this is very difficult to do for reasons you are familiar with.

At any rate, I make this point because there is an awfully lot of money wasted in longitudinal studies that are lost and forgotten.

Meanwhile, as a result of the interest in the possibility of hereditary susceptibility to alcoholism, the chemists have been at work using animal models, and studying people where they can, and have been pursuing various theories as to how alcoholism might be transmitted in the brain, and they have to go to the brain. We are assuming it is not transmitted in the liver or the testicles, but transmitted in the brain.

So there is much more work of this type being carried out now than there was 10 years ago, and also the technology available for carrying out such work has improved vastly and continues to improve in the last 10 years.

And two promising leads—which I will take no more than 2 minutes to discuss because I think maybe somebody will discuss them—one of them has to do with the so-called neurotransmitters, these chemical messengers in the brain, one of which is serotonin. There is a going theory right now that serotonin is involved in drinking behavior. This theory is particularly interesting and tantalizing because there are drugs on the market, which have just come out and are now legal prescription drugs, which influence the activity of this particular chemical in the brain called serotonin. There are already preliminary studies—a couple of studies—that indicate that these drugs may influence drinking behavior, that they may actually reduce drinking behavior.

These are still too preliminary, too early to make any sweeping statements for them, but it has got a lot of us interested.

Chairman GLENN. Reduce what? Reduce the desire for alcohol? Or does it make a person sick?

Dr. GOODWIN. It does not make them sick. It is not like Antabuse. It does not make them sick. The need to drink seems to be reduced.

Chairman GLENN. The desire to drink?

Dr. GOODWIN. The desire to drink. Just anecdotally, I have a couple of patients taking this brand new drug—it has been out one month—who have alcohol problems and after a drink they will say, I do not need another drink; I just do not feel like having another drink. They are not sick, it is just the need to drink seems to be reduced.

Now, this is so early——

Chairman GLENN. If they are thinking this, will they not even take the first drink, if they have been taking this?

Dr. GOODWIN. Well, this will happen. I have a patient who is a reporter who is on the wagon most of the time and was having a virgin margarita—a margarita without any alcohol in it—with a friend having a real margarita, and by accident the bartender put some alcohol in the virgin margarita and the patient picked it up immediately, tasted the alcohol, and ordinarily, having followed this patient for 10 years, I know what his response would have been and that would have been to say, to hell with it, I will just go ahead and drink this margarita. But instead, he said, well, who needs it?

Now, believe me, based on one or two cases, this should not be in anyway blown up into something new or great for alcoholism. But there is one formal study out of the Addiction Foundation in Toronto and there are now a couple of animal studies where animals drink less given these drugs that affect the levels of serotonin. So the serotonin story may not work, but it should be pursued and there should be investments in it.

The drug company that makes the drugs are investing in it. They are based on the so-called deficiency hypothesis. There are two kinds of going theories with regard to the chemical basis for the inheritance of alcoholism. One is the deficiency hypothesis, that a person is born with too little something and then the alcohol corrects the deficiency and, therefore, alcohol becomes more reinforcing for that person.

The other theory follows the notion that some people overproduce something and that this something that they overproduce increases their susceptibility to addiction. I have mentioned the deficiency hypothesis when I talk about serotonin, which I would rather call chemical X than serotonin, actually, because——

Chairman GLENN. Serotonin, is that on the market now?

Dr. GOODWIN. Yes.

Chairman GLENN. Is it prescription or——

Dr. GOODWIN. Prescription drug, right. It is prescribed for depression but it—the serotonin theory has been around for a long time. It was developed at the NIMH here and the NIAAA and by investigators around the country. But we did not know what to do with it exactly except study animals, which is fine, but now for the first time we have prescription drugs—one, so far, but there are another half a dozen on the horizon—prescription drugs which will increase the activity of serotonin in the brain.

Eli Lilly, which makes this particular drug, is sponsoring studies now, testing them out. There are a number of other possible substances, however. As we learn more about what alcohol does in the brain there are more and more candidates for accomplishing the same end; namely, not to make a person sick if they drank, but to just take away at least somewhat the desire to drink. And this is the type of research that should be pursued.

The second kind of research has to do with the overproduction of something. Some years ago it was found that alcohol produces tiny traces of morphine in the brain, which was news to everybody. And so the idea caught hold that maybe alcoholics are really morphine addicts, which did not make a lot of sense, but nevertheless it was interesting. And since then there have been a number of studies that indicate that these morphine like substances that alcohol produces change animals' drinking behavior and also may be overproduced by alcoholics.

Now, that is a long shot. There is a lot of disagreement about how far it should be pursued. My own opinion is that it should be pursued. I think that when we talk about heredity we are talking about the brain and we are talking about chemistry and there are viable theories going. And these theories could have a payoff even before we know the cause of alcoholism. So, as a sort of plug, let me make a plug for this type of research, as well as continuing longitudinal studies until they are finished.

Chairman GLENN. Is NIAAA looking into that?

Dr. GOODWIN. Sure. I might say one thing about NIAAA. It is a very fine Institute. It is governed by the peer review system and sometimes the peers are more sympathetic to some types of research than other types of research and there is nothing you can do about that. And sometimes peers take a dim view of particular ideas and there is no way to keep politics out of anything, including peer review, but I do not have any answer to that. I mean, I do not want to do away with peer review.

Let me just finish with one point and then you can talk about women and alcoholics, which probably will be more interesting. And that is, it was noticed some years ago that not all alcoholism runs in families. Some alcoholics do not have a family history of alcoholism. They have a large family but no alcoholics. So the idea came about that perhaps there were two kinds of alcoholism. There was the kind that ran in families and the kind that did not. And the idea came along that why not compare these two types. So this has been done in a number of studies and it has resulted in the use of terms such as familial alcoholism versus non-familial alcoholism, and there are probably 40 or 50 papers now published on this, based on the possibility that these may be different in some way or another.

It turns out they are different, apparently. One of them is the familial alcoholism. This is the alcoholism where the person has not only one but usually two or more alcoholics in the family, if he has a large family, and this alcoholism begins early. That is the most consistent of all findings, that the alcoholism begins early. You go into a treatment facility for alcoholism and the youngest of the patients will be the ones with the family history, invariably.

Second, the familial alcoholism is a particularly severe, rapidly developing type of alcoholism. Thirdly, the familial alcoholic, based on some information, but not an entirely consistent body of information, that the familial alcoholic is what might be called a primary alcoholic in the sense that there is nothing wrong with them except they drink too much, and if they stop drinking too much, you are no more likely to find any other psychiatric problem than you will in people who do not drink too much.

The message there for treatment is—and this is still more of a hypothesis than a demonstrated fact—the implication for treatment is that if the alcoholic that you are treating does not have a family history of alcoholism you are more likely to find that there is something else wrong with him. And the most likely things you are to find wrong with him is depression, is one. And even more likely, one of the anxiety disorders.

So that you have perhaps as many as a third of hospitalized alcoholics that we now know—because we just started asking—that we now know had experienced serious anxiety disorders—phobia-type disorders, social phobias, particularly, and agoraphobia in women—which seems to have led them into drinking and it was the path to drinking, so to speak, and you do not find this in the familial alcoholics nearly to the extent that you find it in the non-familial.

So once you find out from the patient, do you have a family history of alcoholism? Then the next question is, the next thing to do is see if there is anything else wrong with him. And when we are talking about treatment and prevention, I think this is an important point, although not completely proven by all of the evidence.

So, just to finish, you may or may not hear—you know, there are two types of people in the world; there is the type of people who think there are two types of people in the world and then there is the other type. And we have a lot of people, including the Supreme Court, who think there are two types of alcoholics. At any rate——

Chairman GLENN. Well, stop right there just one second. Did you agree with the Court decision or do you think they were off base?

Dr. GOODWIN. No. They were off, way, way off. They were 50 years out of date, maybe a hundred.

Chairman GLENN. Open the window and let them hear that across the street over here.

Dr. GOODWIN. Oh, boy. They couldn't have been further off.

Chairman GLENN. Do you agree with that, Dr. Lex?

Dr. LEX. No.

Dr. GOODWIN. The other two-typer—I am a two-typer myself. I am a familial versus non-familial. That is where I stand right now. There is another two-typer around from St. Louis named Bob Cloninger who has made popular Types I and II, which I find hard to remember when they are named I and II. But nevertheless, I have decided that Type II is really familial alcoholism with a less easy way to remember it. So in my own opinion, there are two types of alcoholism, provisionally speaking, and that there is a consensus about this but it is just that we give them different names.

I think I will stop there. Thank you.

Chairman GLENN. Thank you, Dr. Goodwin. Thank you very much. We will get to more questions here later.

Dr. Lex, welcome this morning. Dr. Barbara Lex with McLean Hospital, Belmont, Massachusetts. Dr. Lex, welcome and we welcome your statement.

TESTIMONY OF BARBARA LEX, PH.D., MCLEAN HOSPITAL, BELMONT, MA [1]

Dr. LEX. Thank you, Mr. Chairman.

Dr. Goodwin has crosscut a number of the things that I am going to say, but perhaps some of them do bear repeating.

For the past 7 years the research group with which I am associated at McLean Hospital and Harvard Medical School has been looking at substance abuse problems in women. For the past 3 years my work has compared sensitivity to alcohol in women with and without a family history of alcoholism.

Before summarizing my findings I will present some background information that will help to place this work in a scientific context. Currently it is estimated that about 5 percent of women who drink become alcohol dependent, but about 25 percent of individuals receiving treatment are women.

The information about causal associations between alcohol dependence and predisposing factors in women is not as complete as we might like. It is thought that some of the growth of drinking problems among women may reflect higher rates associated with changing social norms and also may stem from diminished stigma associated with seeking help; also, improved recognition of alcohol problems and expansion of treatment programs. And I will just give a very brief example.

Until a year ago judges in the State of Massachusetts had no recourse but to order women in need of alcoholism and drug abuse treatment—and they often go together—to a holding facility in the State Prison for Women. When an appropriate inpatient program for women requiring civil commitment for treatment opened in Boston, I evaluated the first 20 women who came to that institution. All 20 of them had multiple medical, reproductive, psychological, social, and economic problems.

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[1] See p. 200 for Dr. Lex's prepared statement.

We had inquired about family history of alcoholism as part of our history and case history taking. We discovered that 75 percent of those women had a family history of alcoholism. An additional person did not know her family background. She had been adopted and knew nothing about her biological parents. That is a very high prevalence rate, even though it is a very small group, the neediest women at that time in the State of Massachusetts.

Now, no single characteristic is going to explain or predict alcohol problems and no single study can examine all potentially relevant variables. It is currently believed that alcoholism in women, as in men, results from a combination of behavioral, biological and sociocultural factors. But these factors are so interwoven in the processes promoting and maintaining alcohol dependence that only since the 1970s, and certainly with some of Dr. Goodwin's work, has there been sufficient information to begin separating the potential influence of background factors from the consequences of excessive alcohol intake.

Now, the extent to which psychosocial and biological factors differentially contribute to alcohol dependence is incompletely known, as we have heard, and does require further systematic investigation. One useful strategy for disentangling this complex interweaving of contributory factors is to study the persons at high risk for alcohol abuse.

Studies examining biological vulnerability to alcoholism need to be conducted before the onset can produce physiological, cognitive or emotional changes. For women, our knowledge of the specific characteristics that might predict development of alcohol abuse later in their lives is not as well defined as information about parallel processes in men.

Now, historically alcohol problems in women received very little attention. It was believed that sociocultural factors protected women from alcohol abuse—they were somehow insulated from it—that alcohol dependence was similar in men and women, or that alcoholic women were somehow more deviant, more psychologically disturbed and more difficult to study and to treat.

However, comparisons of family backgrounds show that alcoholic women are more likely than alcoholic men to have had an alcoholic parent, especially their fathers, and that the prevalence of alcoholism in fathers of alcoholic women in treatment may be as high as 61 percent. And more women than men who are treated for alcohol problems report that their mothers, brothers and sisters, or their spouses also have alcohol problems.

Now, the impact of genetic and familial factors, as we have heard, has been amply demonstrated in studies of alcohol problems in male adoptees, and we have heard that there are basically, at this time, two types of alcoholic biological parents that are seen in the family history of alcoholic men. Now, there was study of female adoptees in Denmark that included an insufficient number of alcoholic women for making definite conclusions. But it was suggested that alcoholism in women might have a partial genetic basis.

A later, large study in Sweden showed influence of parental alcohol abuse on adopted-out daughters whose biological fathers had later onset of alcoholism, those who had incurred only minor legal infractions, and fathers who required only minimal alcoholism treatment. Now, these are the group that have been referred to as a milieu-limited or environmental-limited, or Type I alcoholics in males. The lower socioeconomic status of adoptive parents in rearing in rural areas had some association with the alcohol abuse rate.

Now, in contrast, if we wish to look at the daughters of fathers who had a severe early onset of alcohol dependence that was associated with criminal behavior—the male-limited, or Type II alcoholics—their daughters did not show up as having alcohol problems but rather were seen for many more treatments of episodes of headaches, backaches and abdominal distress.

So, while hereditary factors might seem to contribute less to female alcoholism, parental alcoholism nevertheless is potentially important, especially since a recent survey of social drinkers here in the United States found that more women than men reported having alcoholic fathers. It is now recognized that age of onset, drinking patterns, and symptoms of alcohol dependence are different for women and men.

For example, drinking problems in women typically begin 4 to 8 years later but alcohol dependence develops more rapidly, even though women drink less frequently and consume less alcohol, so we have a paradoxical situation. Women report that before coming to treatment they had shorter drinking histories, and fewer episodes of binges, continuous drinking, blackouts, morning drinking and delirium tremens.

Numerous studies associate women's alcohol abuse with gynecological dysfunction, with early menopause, with serious fetal defects, and more rapid development of life-threatening cardiovascular, gastrointestinal, and liver diseases. This phenomenon is sometimes referred to as telescoping, or abbreviation of the course of natural history of the development of alcoholism in women.

It is hypothesized that in daily life individuals use assessments of intoxication as cues to reduce or cease drinking. It is believed that young men with a family history of alcoholism are less affected by moderate doses of alcohol than young men who have no family history of alcoholism. To further the example, if cues to reduce or stop drinking, curtail drinking in some way, are indistinct, or if they are delayed or if they are lacking, then the sons of alcoholic fathers may be predisposed to consume more alcohol, become more tolerant to alcohol effects, to continue to consume alcohol and thus accelerate the process of becoming alcohol dependent.

Carefully conducted studies have found that after two to three drinks men with alcoholic fathers showed less body sway and rated themselves significantly less intoxicated than the sons of controls. But each group had comparable alcohol metabolism and similar alcohol levels in their blood. In other words, given the same dose of alcohol, sons of nonalcoholic fathers and sons of alcoholic fathers showed a difference. The sons of alcoholic fathers showed less sensitivity to that same dose of alcohol.

No comparable studies existed for women before we tested this hypothesis. We are currently studying healthy young women with and without alcoholic fathers. The women are carefully screened, they are matched for education, for height and weight, and they are selected because they drink no more than the equivalent of one beer per day. All women are studied during the same phase of the menstrual cycle. Findings from our early studies show that differential responses to alcohol effects can be identified in the daughters of alcoholic fathers.

In our studies we used the same moderate dose that had shown differential responses in the studies of young men. We calculate the appropriate dose for each woman by weighing her. Then we mix that amount of alcohol with some grapefruit juice, place it in a container that gives off a strong alcohol taste and odor and we then make measurements of responses at specific intervals.

Now, since the studies of young men showed that body sway is affected by sensitivity to alcohol, we designed a special device to measure body sway, and we also additionally tested cognitive changes with a symbol matching test. And I will briefly say that this body sway measurement device is the equivalent of walking a straight line, as when the officer who has pulled someone over to the side of the road, except this is a stationary device and the person has to retain her equilibrium on a platform that can wobble. Those wobbles are translated into electronic messages to the computer, and then they are analyzed.

To date our results show that levels of alcohol in blood and the rate of alcohol metabolism are comparable for women with and without alcoholic fathers. We do not see any differences. More importantly, like sons of alcoholic fathers, the daughters of alcoholic fathers sway less after drinking alcohol. In other words, they would be better able to negotiate walking that white line.

However, in contrast to findings for men after drinking the same amount, the intoxication scores for women in both groups were similar. And women's ratings of alcohol effects were lower, and the daughters of the alcoholic fathers out-performed the daughters of nonalcoholic fathers on the symbol matching test, a test of cognitive ability.

Now, one explanation for the apparent differences between the two series of studies might be that results simply come from individual differences. But comparison of individuals with and without a family history of alcoholism from most of the laboratories that are doing this work generally indicate a differential sensitivity. Studies of differences in prepubertal and adolescent children of alcoholics and controls suggest changes in the central nervous system, and some results in youngsters are in the opposite direction of findings from the young adults who are being tested in laboratories after they have been social drinkers for several years.

It is possible, however, to reconcile these seemingly disparate findings by hypothesizing that despite similar patterns of social drinking in young adulthood, greater sensitivity to the effects of alcohol may lead to greater tolerance for alcohol in persons who have a family history of alcoholism.

So, we are wondering if it is not the case that simply being exposed to ordinary social drinking in a social context throughout high school and college or early adult years might accelerate tolerance in individuals with a family history of alcoholism.

Now, differences between men and women also may affect the results. To the best of our knowledge, studies of male/female differences have not as yet controlled for family history of alcoholism. It is very interesting that Dr. Goodwin points out how this information has been around for quite some time but it simply has not been factored into a sample selection of individuals who are studied in the laboratory for male/female differences.

Although our information is still limited, women may generally use different cues to monitor their level of intoxication. In comparison to young men, women have been reported to experience more anxiety associated with alcohol intake and it is fair to say that women in general have different life experiences associated with alcohol, including possible self-medication to relieve mood changes, and physical discomfort associated with disorders of the reproductive system.

Recent research seems to indicate that alcohol interferes with the balance of cofactors that are necessary for the metabolism of reproductive hormones such as estradiol. Clearly, the effects of female gender and family history of alcoholism on alcohol expectancies and intoxication ratings require additional information through carefully designed studies with sufficiently large samples of women who have clearly defined characteristics.

I will do a little summing up, now. We will continue to need studies that give careful assessment to a number of physiological, subjective and behavioral measures. There is one concern that comes to my mind from this brief review I have given you and my reflection on the various relevant findings.

First of all, for some groups the sample sizes are small and they, therefore, may yield results influenced by uncontrolled biases. Larger samples simply generate more stable statistics. Further, if the findings are then to be replicated in another laboratory, the sample selection must be guided by controlling for intervening variables, and the one that we control for is the menstrual cycle phase of the women we are testing. We make sure through assays of hormones that they are correlated with a specific time in their cycle so that we are not taking measurements that may be influenced by random noise in the hormone system.

And, it will be necessary to control for menstrual cycle effects in women until there is evidence to the contrary. The tradeoff factor, however, is that all of this careful screening, matching and controlling for possible biases will inevitably prolong data collection until a sufficiently large sample of individuals can be studied.

This means that it is important to examine several factors in one study, and this means that we would hope that the various laboratories would begin to converge on a consensus of which variables might be most productive to examine. Again, as Dr. Goodwin has said, sometimes there are not funds available to conduct the studies in the most efficient or cost effective way, which sometimes involves collecting data over longer periods of time.

Other factors that contribute to vulnerability, such as family environment and societal trends, may favor or discourage increased consumption. At this juncture, possibly somewhere between 40 and 70 percent of the sons of alcoholic fathers can be definitely classified by a profile of behavioral and biological markers. So we may have, for the case of men, three or four markers when examined that at least reduce some of the chance factor from predicting that there might be an alcoholic outcome if the young man was followed to an appropriate age. We will be able to develop an equivalent profile of markers for women as we continue to evaluate several dimensions of sensitivity to alcohol.

So, thus far our studies have identified body sway as one marker that seems to be fairly robust, and it is something that is interesting to us. We would like to know what that mechanism is that facilitates this differential in body sway in women and men who have a family history of alcoholism. The performance on the symbol matching test was interesting to us, also. We are currently conducting studies using more complex cognitive studies to see whether or not these tests can indicate some location in the nervous system that may be influencing the predisposition to develop alcohol problems. Yet, we have to hone in on this using, perhaps, some very blunt instruments at the present time.

We foresee that the data will continue to need interpretation using models that include by psychosocial and biological contributions to the mechanisms of inheritance and transmission of vulnerability. One might add that if individuals are raised in an environment where no alcohol is available, they might certainly have the predisposition but if the alcohol is not available through whatever measures—if one were to go to one of the Islamic Nations like Saudi Arabia, for example, that individual might have a very strong proclivity to become alcoholic but the alcohol simply would not be available. What we do not know is just exactly what would happen to that individual, whether something else would happen or whether this process would simply be aborted.

I carry, too, the same message, that we need sufficient resources to continue our investigations of these other factors that are involved in vulnerability to alcoholism and we also need appropriate models for education and intervention and treatment. I might mention that within the last decade the concerns about children—and these are children of all ages—affected by parental alcoholism have come to public attention. There have been special research and policy task forces and commissions that have convened to look at the magnitude of these problems. NIAAA has been involved in some of those, and it is estimated by some that 28 million Americans have at least one alcoholic parent.

As yet, however, there is not a clear consensus among clinicians and scientists and educators about the type, extent or severity of possible impaired functioning of children of alcoholic parents. What I am trying to say here is very simple. It may even be reductive. But not every individual who carries a risk for becoming alcoholic, familial alcoholism is going to develop into an individual with alcohol dependence. There may be intervening variables. It may be that in a high school situation a caring teacher or guidance counselor, an education program, something of this nature, the school nurse, something as simple as at that level. If the family becomes known to the Department of Social Services in a locality, again, the individuals involved in delivering care to those people should be aware of the fact that alcohol and also substance abuse are important risk factors.

In Massachusetts I am told that there are something like say 8,000 youngsters in foster care and between 80 to 90 percent of those youngsters who are looked after by the Department of Social Services have a background of alcoholism or substance abuse in their household. There is an enormous amount of money being expended right now for foster care, group home treatment, therapy, other interventions that need to take place.

It is also the case that there is a thread throughout what I have presented, that women's problems seem to show up affecting their reproductive systems, among other bodily organ systems. Many women do not have a primary care physician. The family doctor model has been diluted over time. But women do seek out gynecological care on a more routine basis.

Now, I just want to quote a couple of statistics and then I will close. The statistics from which I am quoting are not studies that we did all were also done at one of the hospitals in the Boston area. The general medical service examined groups of women who were suburban dwellers, middle class, whatever label one wishes to choose, in the fee-for-service sector. They were not clinic patients. They were not the poorest people in the metropolitan area.

In a practice that simply screened women seeking gynecological care who were getting routine testing or coming in to see if they were going to be bearing children or whatever, the rate was 12 percent who answered the disclosing factors on a questionnaire designed to identify alcohol problems. This is just 12 percent of women coming in for a routine pap test or something of this nature.

In a practice that looked at individuals coming for help with premenstrual syndrome, 21 percent of the women—1 out of 5—when they answered their screening questionnaires about their general health—there were alcohol related questions embedded in that questionnaire—one out of five individuals acknowledged that they had some symptom of alcohol dependence. Perhaps they were drinking excessively before going out to a social engagement, drinking in the morning, drinking when other people were not around, and so forth.

So, one area that might be targeted in the health care sector, for women especially, would be the OB-GYN practices and clinics that are screening for public health purposes using various tests on the reproductive system.

Thank you.

Chairman GLENN. Thank you, Dr. Lex.

Dr. Goodwin, how responsive to treatment are the familial versus the non-familial alcoholics? Are there certain kinds of treatment that will work better with one or the other type?

Dr. GOODWIN. The studies, to the extent there are any, is that the familial are more resistant to treatment. As I mentioned, it appears to be a more severe form of the illness and that they are more resistant to treatment. I do not think, in the case of familial alcoholics, there is any other treatment than total and complete abstinence.

There are some people who have been playing around with controlled drinking for alcoholics and—this is a very unpopular thing to be doing but they do it anyway—but they will concede that in the case of familial alcoholics they have given up, that it is a poison for the familial alcoholic and that is all there is to it, whereas in the non-familial alcoholic, perhaps the—in other countries, I might add, other than the United States, the goal of moderation of drinking as opposed to total abstinence is conceived of as an acceptable and appropriate goal.

Chairman GLENN. Are there any other approaches besides the Alcoholics Anonymous approach, the STEPS and so on? Are there other approaches that are that successful or that you know about, or is that the best?

Dr. GOODWIN. Well, we do not really know. We do not have studies where patients are randomly assigned to different kinds of treatments and we do not know what the untreated alcoholics do. We do know that there is a pretty high spontaneous remission rate in alcoholics. There are an awful lot of alcoholics who stopped being alcoholics without professional care. This was not known until fairly recently, but I think everybody agrees on that.

The predominant mode of treatment in this country has been built around the AA model but this has not been true in other countries and there is sort of a transatlantic controversy going on as to whether AA applies to more than just a small minority of alcoholics and a certain amount of evidence, in fact, from England that a very brief intervention achieves as much as lengthy expensive intervention when it comes to alcoholism, that if the person is ready to stop, that is always the crucial thing, if the person is ready to stop.

So, yes, there are other modes but, again, we do not have the kinds of studies with random assignment, again, to different treatments that would permit us to say that any of the treatments are superior to no treatment. There is a general belief that they are, and I certainly believe in treating alcoholism. But if you want hard evidence that the natural history of the disease is not as pervasive a factor in the recovery as intervention, you will not find data of that sort.

Chairman GLENN. Dr. Lex, you mentioned that 5 percent of the women who drink become alcoholics, 25 percent of the persons who receive treatment for alcoholism are women, or female. What are the comparable figures for men? What percent of men who drink become alcoholics? Do we have that?

Dr. LEX. The proportion of men to women is variously estimated anywhere from 4 to 1, 5 to 1, and it depends on the——

Chairman GLENN. Are those the ones receiving treatment? Because if you took your figures, 25 percent of the persons who received treatment for alcoholism are female, that means 75 percent are men.

Dr. LEX. Right.

Chairman GLENN. But what I was dealing with was is there a different percentage of men who become alcoholic as a percent of those who drink? Your figures were that 25 percent of the women receive treatment, yet only 5 percent of the women who drink become alcoholic. What are the comparable figures for men? Do we have that?

Dr. LEX. In terms of individuals seeking treatment? No. At this point, to be honest with you, I have not looked at that.

Chairman GLENN. Well, it would be 75 percent of those receiving treatment, but what I was getting at, of the total of men, what is the comparable figure to your 5 percent of women becoming alcoholics?

Dr. LEX. Let me say this, that the numbers are not cast in concrete. It is very difficult to ascertain just who is alcoholic. If one does a household survey, for example, one may get under-reporting.

The data we have available come from different kinds of treatment facilities so that in some treatment facilities there may be no women admitted whatsoever and others may cater to that particular clientele. What I am trying to convey is that the proportion of individuals who come to treatment because of the medical consequences, perhaps, or because of the social consequences, is proportionately higher than one might predict.

Chairman GLENN. Dr. Goodwin, do you have any figures on that? What percent of men who drink become alcoholics that would be a comparable figure?

Dr. GOODWIN. Well, these figures are made up by administrators, generally, in order to influence Congress in one way or another and we——

Chairman GLENN. Obviously, they impressed me or I would not have asked it.

Dr. GOODWIN. Well, I use the word "ball park" a lot, you know. That word gets thrown around a lot. The fact is, when you go out and do a household survey of alcoholism, the alcoholics are not home, as a rule. But also, the definition of alcoholism becomes absolutely crucial, and you will notice that the Government will increase its figures if—I mean the administrators—will increase the figures by including a group called "problem drinkers," alcoholics and problem drinkers. And this gives it an infinite contractibility or expansibility by throwing in other groups.

Also, it might be interesting to you to know that since Dr. Lex's colleague, Dr. Mendelson, was head of the Alcohol Institute here in the late 1960s, there were estimated to be 5 million alcoholics. Now the last official estimate I saw was 14 million alcoholics, which includes problem drinkers. But it also happens to be proportionate with the amount of spending that went into the field, the increase, the increase in alcoholics and the amount of spending seemed to go in parallel.

At any rate, to answer your question the best that I can, having looked at the figures and thought about it, I think that the best figure, in my opinion, is that about 1 percent of women in the general population become alcoholic and about 5 or 6 percent of men in the general population become alcoholic. Those are the figures I use. I will not swear by them.

Chairman GLENN. Five percent just general population without regard to whether they have whatever parents?

Dr. GOODWIN. That is over 16. I am talking about over 16.

Chairman GLENN. Regrettably, we are going to have to move along here. There are a lot of questions that I have here that I would like to get into and we may submit those to you for a reply, if you would, so they could be included in our record here. We do have two other panels we have to move along with here this morning.

I appreciate both of you being here. Thank you very much. It was very interesting.

Our next panel deals with behavioral and environmental determinants. Dr. Robin Room of the Alcohol Research Group, Medical Research Institute, Berkeley, California, and Dr. George Vaillant, Dartmouth University, Hanover, New Hampshire.

Thank you. Dr. Room, if you would proceed, we would appreciate it.

TESTIMONY OF ROBIN ROOM, Ph.D., ALCOHOL RESEARCH, GROUP, MEDICAL RESEARCH INSTITUTE, BERKELEY, CA [1]

Dr. ROOM. I am going to skip some of what was written in the testimony on our research and on the social location of problem drinking. But I would add something in light of, the colloquy that just went on. We are engaged in general population studies of drinking patterns and drinking problems. It is very easy for those of us who are in the field to get cynical about these figures, but there are some realities behind them.

In estimates from our surveys, something like 3.4 percent of the American adult population reports it has had some treatment for its own alcohol problems, including Alcoholics Anonymous, in their lifetime. For treatment within the last year, it would be a little over 1 percent. That pretty well matches up with the figures that you can get from looking at treatment system data.

Chairman GLENN. 3.4 percent now have had some treatment for alcoholism?

Dr. ROOM. For alcohol problems in their lifetime. And that includes going to Alcoholics Anonymous. It may not be formal treatment by a professional.

A much larger proportion than that have gotten into a scrape with alcohol in the course of their lifetime. Probably about 80 percent of the population never drinks enough to get into much trouble with drinking. A third of the adults in the United States do not drink at all. Only a half of the adults in the United States drink as often as once a month.

So, when we strip it down to the people who ever drink enough in their lifetime to get into any substantial problem with alcohol, then we are at the most talking about 20 percent of the adult population. If we ask about people who have gotten into a scrape within the last year—and I am using that word to make it clear that I am not necessarily talking about something that people would diagnose clinically as alcohol dependence or alcoholism—then we are down to more in the order of magnitude of 10 percent.

When you are talking about people who would fit those clinical signs of alcoholism, then you are getting down into the kinds of figures that Dr. Goodwin was quoting in the general population; 5 or 6 percent of the men, maybe, and 1 or 2 percent of the women. Heavy drinking is very much a male phenomenon. Women are only about 25 percent of the treatment population. For an equal amount of drinking, a woman is at least as likely to get treatment as a man is. Women in our society are generally better users of the health care system than men are and this seems to be becoming true as much for alcoholism treatment as it is for other aspects of the health care system.

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[1] See p. 231 for Dr. Room's prepared statement.

I hope that I have been responsive to the questions that you have about the numbers.

I am also going to omit from my testimony some specific recommendations that you will find at the end of it that reflect the fact that alcohol and alcohol problems range very widely in our society and there are a very large number of governmental agencies that in one way or another have some jurisdiction or responsibility over alcohol problems. There are a lot of problems that fall between the cracks, given the current arrangement, both at the Federal and at State and local governmental levels. So that there is a very substantial opportunity for doing something to prevent and manage alcohol problems better by coordinating the activities between different agencies of the Government in this area, and you will find some specific recommendations on that at the end of the testimony.

What you asked me to talk about, though, was to focus on environmental and behavioral risk factors in alcohol problems and on the interaction of these factors with genetic and other factors, and this is a large topic, obviously, that I can only tackle selectively. And what I was going to focus on for you today was on the issue of change, how changes happen and how large the changes can be in drinking patterns and drinking problems in a given population.

Some of the most interesting work in recent years has involved opening up this dimension of time and change to systematic study. It has involved work in a number of research traditions. For instance, time series analyses of health and social statistics, primary source work in social and cultural history, and analyses of trends in population survey data.

While much of the cross-temporal research has been focused on the understanding of marginal changes of what happens when you raise the price of alcohol by 5 cents, it has also underlined the fact that big changes do happen, big changes in the level of drinking in the whole population and in the rates of alcohol-related problems in the population.

One of the truly dramatic changes in world history came in the 1830s and the 1840s in the United States with the first wave of temperance sentiment in the U.S. Those who have written the testimony can see that in figure 1 [1] the estimated level of per capita consumption in the U.S. fell by a half in the 1830s and the 1840s. Then consumption went back up somewhat during the Civil War and again at the turn of the century.

Many commentators, both those at the time and more recent commentators, focus on this backsliding, concluding that moral suasion in the early temperance movement did not work. But in the longer perspective, there was a lasting change. The per capita consumption in the United States has never since reached the levels at which people drank when the republic was young.

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[1] See p. 237.

There are a couple of other examples of big changes that I have brought some charts for you on. Cirrhosis mortality fell very dramatically in Paris, a heavy wine drinking city, during the two world wars, as alcohol became increasingly unavailable to the population and in the situations of privation in those two times.

In Finland, on the other hand, you can see what happened with the liberation, as they called it, of beer into the grocery stores in 1969; a very sharp increase in consumption and along with that—you can see this in the tables,[1] number 1 and 2—a sharp increase in alcohol problems across the board; social problems, casualty problems and physical health problems associated with drinking.

As the consumption of alcoholic beverages rose, so did the rates of most alcohol related problems. You can see the effect in the opposite direction for Poland, looking at figure 4[2] and at the table at the end, table 3.[3] Alcohol consumption rose to a peak in the late 1970's and then declined in a period of heightened morale in the Solidarity period—one of the original 10 demands of Solidarity was that the Government stop pouring alcohol down the throats of the people—and later, under the restrictions of the martial law period when there was a very severe rationing on alcohol.

As the Finnish and Polish examples demonstrate, one clear finding in these studies is that changes in the level of alcohol-related problems tend to follow changes in the level of consumption in a given population. This is true particularly for the physical effects of sustained heavy drinking such as liver cirrhosis and head and neck cancers, but it tends to be true also for other alcohol problem indicators such as rates of casualties and of violent crimes.

There are a few studies which directly measure changes in the drinking patterns and problems of individuals in response to such factors as a price increase or an economic downturn. We need more of those specific individual level studies. It is often intuitively assumed that very heavy drinkers would be less responsive to such factors than lighter drinkers, but that is not how the data comes out. They are at least as responsive proportionately, and that means in absolute terms they are more responsive to, for instance, an increase in price than lighter drinkers are.

We can thus conclude that extraordinarily large changes in drinking patterns have occurred in particular populations, sometimes in a relatively short period of time. In a particular population where we are holding roughly constant the genetic composition or susceptibility, therefore, the manifestations of heavy drinking and of drinking-related problems can vary widely. Furthermore, changes in the drinking levels and the patterns of the population as a whole seem to affect the behavior of those who are presumably most genetically susceptible; that is, the heavy and the problem drinkers.

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[1] See p. 239.
[2] See p. 238.
[3] See p. 240.

For drinking behavior and problems, as for human behavior generally, the constraints of our individual genetic composition seem to allow for a very wide behavioral repertoire.

What does this imply for our efforts to reduce the rate of alcohol related problems in our society? One implication is that we need to understand the conditions under which changes, big and little, occur. To understand the specific genetic mechanisms of susceptibility to or protection from heavy drinking or alcohol problems is an exciting and fruitful scientific task, but the individual testing and counseling that this makes possible may be ineffective if it goes against fashionable behavior.

Down at the end of my street these days there is a billboard from an airline which shows a woman apparently of northern European ancestry sunbathing on a Hawaiian beach; it has the message, "Bake 'til golden brown." Now, we can tell this woman that the genetically determined color of her skin means that she has a heightened susceptibility to skin cancer. She is risking a disease by baking until golden brown, and genetic factors are certainly involved in that. But until our society changes its ideas about the body beautiful, the airline can still count on a positive response to its invitation of a behavior which is for the genetically susceptible a health risk.

What we have learned about how changes in levels and patterns of drinking occur is mostly by studying natural experiments of what happens. Natural experiments, I would add, are what social scientists use to describe what legislators do very often. In other words, it simply means where a society is making decisions that are not being determined by a research design. And as for other commodities, alcohol consumers are somewhat responsive to price and to other limitations of availability.

In societies like our own, the main way in which big changes in habitual behaviors happen is through movements seeking social change or spiritual renewal. When de Tocqucville wrote of Americans as a nation of joiners, what he had observed was the first flush of the temperance movement in the 1830's.

Today we find ourselves in another era of growing social ferment over alcohol issues. The most visible signs of this ferment have been political movements which aim to change someone else's drinking, groups like "Mothers Against Drunk Driving" and "Remove Intoxicated Drivers." But there have also been quieter changes, changes in people's hearts about their own behavior, as many have switched to low alcohol beverages and then to no-alcohol beverages, as alcohol treatment capacity has grown, and as movements of self-help and of mutual help have grown by leaps and bounds. Alongside the burgeoning fellowship of Alcoholic Anonymous, a whole new many-sided movement of Adult Children of Alcoholics has grown up in the space of less than 5 years.

In my view, what we are witnessing is the unraveling of a half century old consensus about alcohol issues in our society. The college kids of the late 1920s and the 1930s, roughly speaking, that is, the people of President Reagan's generation, staged a partly successful generational rebellion against what they defined as Victorian morality.

Growing up during prohibition, many students took up drinking and, indeed, heavy drinking as a signal of generational protest and of personal autonomy. Fifteen or 20 years later, with their heavy drinking now becoming a problem in their life, people from that same wet generation swelled the ranks of Alcoholics Anonymous as it grew in the 1940's and the 1950's. Today their daughters and their sons, now in the 30's or 40's, from the core of the Adult Children of Alcoholics movement.

The college kids of the 1920's and of the 1930's won their battle against prohibition. In the wake of repeal, alcohol problems turned back from being a public issue to being a private anguish. To be thought at all cosmopolitan or forward thinking, one could not utter a word against drinking. Eventually, in the 1940's, an effort was started to provide for more humane treatment for the growing numbers of alcoholics. Those in this nascent alcoholism movement had to reassure everyone that they have nothing to do with temperance and were not opposed to social drinking.

So this was the political and cultural environment in which the idea took root that there were two types of drinkers—social drinkers and alcoholics—that were marked off from each other at birth or at an early age. Such a view made it possible to provide humane help while not threatening or involving the rest of us social drinkers.

Meanwhile the idea that drinking was a part of everyday adult middle class life continued into the post-war years of growing prosperity. We did not notice in the 1960's and 1970's, when everyone was paying so much attention to illicit drugs, that a new generation of college students were part of a steep rise in alcohol consumption, that the consumption went up by 30 percent in a decade in the same period.

Fifteen or 20 years down the road, the heaviest drinkers of this college generation are now providing a new flood of recruits into Alcoholics Anonymous and alcohol treatment programs. These generational experiences, in my view, are an important part in the ongoing shift in the cultural position of alcohol in our society. At a more general level, it appears that we are just beyond the crest of one of the long waves of alcohol consumption that researchers have noticed in many industrial societies.

The popular movements which we see around us, as well as the pressure for legislative restrictions on drinking, seem to me part of a self-corrective process with which a society like ours comes to grips, however belatedly, with the real social and health costs of levels of consumptions which we used to find tolerable.

Chairman GLENN. Thank you very much, Dr. Room.

Dr. Vaillant.

TESTIMONY OF GEORGE VAILLANT, M.D., DARTMOUTH COLLEGE, HANOVER, NH

Dr. VAILLANT. Thank you.

I think I can serve this group the best if you will follow my pictures in my remarks rather than any of my words, and if I try to summarize my perhaps unique privilege in being allowed to follow children into late middle life with and without the disease of alcoholism through longitudinal studies.

Making sense out of alcoholism is a little bit like learning how to pat your head and rub your stomach at the same time—you have to be able to conceptualize paradoxes. And I want to try to illustrate four paradoxes today. One, that alcoholism is a disease, but a disease that arises from bad habits. Two, that alcoholism, although it has a significant genetic etiology, is one whose prevention will probably be most effectively achieved through social and economic means.

Third, alcoholism, if not cured, is very often a fatal disease, but that hospital intervention plays only a small part in recovery. And finally, although alcoholism is a very painful disease, Mr. Deaver's best assistance in recovery is probably not a good defense lawyer.

Turning to the first question, if alcoholism was a nice tidy disease you would expect nonalcoholics to drink a small amount and alcoholics to drink a large amount, so if you looked at the distribution of drinking in a population, there would be a two-humped curve. If you look at the first figure,[1] alcoholism is distributed in an even curve throughout the population with, admittedly, 10 percent of the population drinking 50 percent of the alcohol. And not surprisingly, it is that 10 percent that has most of the problems.

But consider heart disease as an analogy, that arteriosclerosis is distributed on a smooth curve throughout the people in this room and throughout the country, and it is the people with the most arteriosclerosis that suddenly start having problems. But they do not start having problems the way pregnant people start having predictable problems. They start having problems on the basis of increased risk. The more arteriosclerosis you have, the more likely you are to have problems that are called heart disease.

So perhaps the genetic defect in alcoholism is like having a car that genetically has no seat belts. Disaster is not assured. It is simply that the risk goes up.

If you look at the second picture, on the left-hand column you can see a distribution of lifetime alcohol problems, and in the second column, the distribution of 400 inner city men followed for 30 years. Now, here the rate of alcoholism is going to be higher than those quoted by Dr. Goodwin and Dr. Room because I am talking about lifetime risk of problems rather than problems at one point in time.

But you can see that once an individual admits that he has problems controlling his drinking, then the likelihood of his having an awful lot of problems is greatly increased. And the difference between that person who has difficulty with control and that person who someone in the community identifies as alcoholic is again one of risk. Not everybody who has trouble controlling their drinking and who has many problems in drinking will ever be identified as having a risk of alcoholism.

But clearly, what Dr. Room has said, to the extent that you affect alcohol consumption, you are going to affect the rate of alcohol related problems and you are going to affect the rate of alcoholism.

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[1] The illustrations referred to throughout follow Dr. Vaillant's prepared statement.

In Japan, in many parts of Japan there are no coronary care units. They do not have significant coronary heart disease because they do not have the bad habits of too much salt, too many cigarettes, too many animal fats and not enough exercise. When those Japanese move to America and are exposed to our bad habits, they then develop heart disease. A disease of alcoholism is the same way, that genetics only increases risk and there are many other variables that also affect risk.

In turning to the third slide and the second paradox of how do we affect risk, we know that adult alcoholics are lower social class, more unemployed and have more mental illness than nonalcoholics, and we assume a causal etiology, which is incorrect. It is not depression and unhappy childhoods that cause alcoholism, statistically. It is alcoholism that causes unhappy childhoods and causes depression and mental illness. Therefore, if you look at the bottom four rows of the third diagram I have handed out, you can see that the childhood of these social drinkers and alcohol dependent men did not show lower social class, did not show more multi-problem family membership and did not show more emotional problems.

In turning to the next slide, of 400 10-year-olds followed to age 50, there were 51 men who came from alcoholic families but which were not multi-problem. That means perhaps, that their father died when they were young, or moved out of the house, that they lived with a nonalcoholic step parent, or their father developed alcoholism after they grew up. But they did not have multi-problem families. They had blood relatives with alcohol; 27 percent of them became alcoholic.

There were 56 men who grew up in clear inner city multi-problem family households but in which neither blood parent was an alcoholic. They might have had an alcoholic step parent. Only 5 percent of them became alcoholic, the same 5-fold risk that Dr. Goodwin describes in terms of risk of alcoholism.

But I want to make a point here in that the studies, talking about two types of alcoholism, familial versus non-familial, do not control for environment. If you grow up in a multi-problem family, to the extent you develop alcoholism, it is more likely to have an early onset and it is more likely to be severe. In other words, it is the environment—the multi-problem family environment, at least in our data—that affected whether it was early onset and whether it was severe. It was the genetics that increased the risk of developing alcoholism at some point during one's life.

If one turns to the next slide, this is an over-simplification, but it makes the point. Over 20 years in the United Kingdom, a 2-fold absolute increase in the price of whiskey affected its consumption by 4-fold. Now, in the United States since 1950, the cost of gasoline has gone up 5 times, people's income has gone up 4 times, the cost of alcohol has gone up twice, and the excise taxes have gone up even less than that.

Chairman GLENN. Could I stop you there and ask a question. Do you have any comparable studies on cheaper alcoholic beverages? Beer, for instance? Whiskey is a little higher priced.

Dr. VAILLANT. That is a good point. It certainly would not be 4-fold. Changing the price of beer only modestly affects the consumption. But if you look at the United States over the last 35 years, the per capita consumption of alcohol has doubled. At the same time in terms of discretionary disposable income, the cost of alcohol has fallen 50 percent. So that if you look at the total alcohol consumed, it is price sensitive. It is not black and white. We are only talking about risk factors here.

But if you change the excise taxes, if you change the cost of alcohol, you only have to think of your own behavior when you are traveling first class on an airplane versus when you are traveling economy. You drink more first class because it does not cost anything. And this is only one variable, but it is going to affect the societal risk, as Dr. Room has addressed in more detailed studies.

Chairman GLENN. So from a policy standpoint here and what you would recommend, it would be easy to say, of course, we put more alcohol excise taxes on and they apply to beer. Beer consumption would go down somewhat. I am not sure it would go down that much. It is a little different though on a dollar beer or whatever beer costs now in a bar as opposed to a bottle of whiskey going from say $12 to $22 or something like that. That is a different problem than a person walking in to a bar for a beer.

Plus, do we have any breakdowns in this general field on what part of our alcohol problem is caused by just beer consumption versus whiskey consumption, for instance? Do alcoholics automatically go to the higher alcoholic content in the whiskeys?

Dr. VAILLANT. Not at all. The only thing that produces a risk of alcohol-related problem is how much alcohol you consume, and it does not matter whether you do that in six shots every 12 hours of Chivas Regal or six 12-ounce cans of the cheapest beer. It is the alcohol that does it and there is no question that at different times and different segments of the population, how you drink that beer is going to be different.

Chairman GLENN. Should our policy be to tax alcoholic content?

Dr. VAILLANT. Very much so, and so that what you have to pay—in the Wall Street Journal today, in order to make more money, Jack Daniels is putting water in each bottle of Jack Daniels so they do not have to pay as much taxes so they can make a bigger profit. If you can reinforce the liquor industry for watering their product, you are simply going to reduce the risk. You are not going to wipe it out. You are only working at risk and it is relative. But what you want to tax is the alcohol. And in the same sense that you can manipulate this through counter-advertising.

One reason that alcoholism in women is going up is that they are a targeted group by the liquor industry. The five leading women's magazines will not accept articles showing the health risk of smoking and alcohol because of the control that the advertising has on their editorial. So that, I mean, we are dealing with social forces and counter advertising is something that the Government can manipulate. Excise taxes are something they can manipulate.

The next figure addresses the same kind of issue in a slightly different way. In this inner city sample, 61 percent of their parents were foreign born. That means we could contrast the boys that had been raised by Italian and Greek parents with those who had been raised by Irish and old American parents. And the Greek and Italian parents tended to make wine in the basement, give it to their children with meals, and if a 25-year-old child comes home drunk, you throw him down the stairs.

In contrast, in the Irish families and in, certainly, American culture, if alcohol touches your lips before you are 21 you can break your mother's heart, and if you are not dead drunk before you are 22 you break your father's heart. So it is a little bit—our national policy towards alcohol is a little bit like forbidding children to learn how to drive and then not having speed limits on the autobahn. It is terribly important to try to change public policy through counter advertising against heavy drinking. It is not modest drinking that is the risk. It is heavy drinking that is at risk.

The next picture is simply following 100 alcoholics 10 years after they leave the hospital, and it simply is a way of dramatizing that in 10 years a third of alcoholics are dead and another third are abstinent. Probably both Senator Glenn and the Surgeon General understates the number of alcohol related deaths. There are probably 200,000 to 300,000—depending on the way you want to bend your figures—a year. Alcohol abuse, if you are a clear alcoholic, your risk of dying goes up 3-fold and 15 years are taken off your remaining lifespan. So it is very often a fatal disease.

But if you look at the next slide, if you take these same 100 alcoholics that are followed for 10 years, they had 1,500 hospitalizations, only 3 percent of which led to abstinence for more than a year. Now, this means that a few people were voting early and were voting often, so I do not mean to say that hospitalization for alcoholism is ineffective. I simply want to underscore the point that the treatment of alcoholism involves relapse prevention; it does not involve getting sober.

Mark Twain said, stopping smoking was so easy he had done it 20 times. One of the signs of alcoholism is that you have been on the wagon for a month. So what we need to do is to shift the proportion of our health spending into imaginative outpatient care, into employment assistance groups and away from trying to encourage people to think of hospitalization as the cure, analogous to diabetes. Hospitalization may be life-saving in diabetes, but it does not change the natural history of the disease. It is community maintenance, it is preventing relapse that is important in diabetes, and it is research and relapse prevention that is going to lead to breakthroughs in alcoholism.

The final point I want to make is simply in reference to the Supreme Court decision. Alcoholism is a disease. It is a painful disease, and yet defense lawyers are not helpful. People with leprosy lose their fingers, they lose their noses, the lose their ears. It is not because the leprosy bacillus eats them away. They lose their capacity to feel pain and then trauma causes the damage.

And the difficulty in treating alcoholics is that they have reduced sensitivity to the consequences of their behavior and therefore if one is too helpful, if one is too good an enabler or defense lawyer, you can protect the alcoholic from hitting bottom, from what Dr. Goodwin called spontaneous remission, and it behooves anybody who has legislative control over being nice to alcoholics because it is a disease—I do not mean being humane, I mean being nice to alcoholics because it is a disease—to go to a few AL-ANON meetings and try to see how loving people have learned to be helpful to alcoholics by not facilitating their disease but allowing the press photographers to take pictures of Wilbur Mills in the Washington fountains, which probably helped facilitate his recovery rather than keeping it out of the newspaper.

Thank you.

Chairman GLENN. Thank you very much, both of you.

We heard from Secretary Bowen yesterday about the activities of HHS in the area of alcohol abuse prevention. His program's primary function or focus is on the public information and education. Do you think that is the most effective way to go at this? Are there other approaches that we should be using also?

Dr. VAILLANT. Yes. I think you certainly have to do much more than education because education is not as effective as advertising and the available techniques for changing how people consume things and buy things. I think that public education in helping individuals, high school students, make decisions about how to drink intelligently, especially if they have alcoholic relatives, to help people make distinctions between going to Hawaii for the sun versus toasting themselves brown and helping the public understand issues of relative risks rather than trying to see things in black and white. But there is good research to show that alcohol education programs on college campuses do not change the rate of fraternity alcohol consumption, where if you specifically teach people how to monitor their alcohol consumption and raise consciousness about how they are drinking, you can reduce alcohol consumption.

So that education is a good thing but it requires research and it has got to be done not by throwing money at the question but by controlled evaluation.

Chairman GLENN. Dr. Room, do you have any other approaches besides that of education?

Dr. ROOM. I think we have to start moving to thinking about alcohol problems as being something for which we need to set an overall policy, and alcohol education and public information should be set within that framework of an overall policy in the same way that we have for other major health problems. When we are dealing with problems of AIDS, when we are dealing with problems of drug abuse, we think in terms of an overall framework of action, perhaps not as much as we should, that is not focused on any one strategy as being a sufficient strategy in its own right.

There is a tendency, I think, in our society to fall back very quickly on the educational strategy. It fits very well into our culture, but if it is going against the cultural tide, it is like spitting into the wind, and—as we saw with that college generation at the end of prohibition—can even be counterproductive if what is seen as an authority figure is laying on with a heavy hand in an educational message.

What we have learned from the smoking area, for instance, is that the most important thing about an educational campaign or effort is that it has to be fitted into a general frame of the shifts in the culture that are going on. The same anti-smoking campaign that is showing an effect now might have had no effects at all 20 years ago, because we have had a big shift in the cultural frame around tobacco smoking. That is the same kind of thinking that we have to do in the alcohol area where any program of alcohol education has to be fitted into a broader frame of action and policy.

Chairman **Glenn**. Just scaring people does not seem to change their habits that much. This may be a far-fetched analogy, but I will make it anyway. Back in years past when I was flying every day in the Marine Corps we had the squadron safety programs, trying to cut down accident rates. And so the big tendency back in those days was you had all the publications with all the blood and gore pictures of accidents and things in it and you were trying to scare people into being safe. I do not know if it made one whit of difference, quite frankly.

Then there were some other approaches tried and they may have been a little more successful. You gave people awards and certificates and there was a positive approach. It was something positive that if they checked the airplane in such and such a way and you really went into a prevention type program from a different approach.

Now, I do not know that that same analogy applies here, but is there any positive approach that has been taken other than just telling people you are going to have diabetes, you are going to have this, you are going to have that, you are going to have heart problems, you are doing this all to yourself, you are going to have an early death, and so they go right ahead and drink anyway? Is there a positive approach on this that has ever been tried?

Dr. **Room**. Yes. There are a variety of different kinds of alcohol problems. If you are going to worry about preventing cirrhosis, then you have very little choice but to start worrying about the overall amount of drinking. But if you are worried about preventing casualties or preventing the social problems from drinking, then one of the major things you can do is affect the insulation around the drinking; that is, make the world safer for drinkers and from drinkers. When the Congress moves towards air bags, air bags in cars are going to affect the casualties for drinking drivers and for those who are run into by drinking drivers at least as much as everyone else.

So that if you start thinking about alcohol problems as going across a very broad range of problems, for many of them we can actually do something about the problem that comes from the drinking without even affecting the drinking. And those kinds of strategies tend to be not paid attention to so much for a couple of reasons.

One of them is because you tend to end up, when you are going to prevent alcohol problems, thinking about doing something about the alcohol. And the second is, there is a kind of moralism that we have in our culture where people start laughing uneasily when you say "make the world safer for drunks." We have a cultural set that the drunk should suffer for his or her behavior. From a public health point of view, that is something that we really need to struggle against, as we begin to think about how we can practically reduce the problems from drinking in a society that has decided that it is going to tolerate quite a lot of drinking.

Chairman GLENN. Yes. I saw yesterday here the new TV spots that Dr. Bowen showed us yesterday on TV here, using sports figures, rock groups and things like that, that there are other kinds of drinking here. There is tomato juice, there is ice tea, and there are a lot of other things here, and why put alcohol into this stuff. And using popular sports figures and rock groups and things and I was quite attracted to them. I thought they might do some good.

It did not just say, you know, you are going to cause a wreck, you are going to do this, you are going to have whatever your physical problem is going to be if you continue the pattern of drinking started in high school or whatever. It says, okay, you are going to drink, but why do you have to drink something with alcohol in it? Make it popular to take off in a little different direction, which is an approach I had not seen before. I thought it was very good.

Dr. VAILLANT. I think you have got exactly the right model with how do you teach Marine flyers, say, flying habits. Clearly you cannot do it by scaring them. But you can do it by affecting costs.

I live on the border between Vermont and New Hampshire and in New Hampshire no one cares if you wear a seat belt. In Vermont the police stop you and we have got a small, well-supervised town. And when I cross the bridge, sometimes, I put on the seat belt. I am educated. I know that seat belts save lives. But the cost is greater in Vermont for not wearing seat belts than it is in New Hampshire.

Chairman GLENN. You obviously do not do it for your own safety. You do it to prevent getting a ticket.

Dr. VAILLANT. I am saying cost. You see, we are talking risk. We are not talking black and white, which is why you cannot scare people. One cigarette is not going to be a coffin nail. What you want to do is to help people evaluate risk in creative ways.

Chairman GLENN. Dr. Room, in your testimony you commented that a restrictive law can offer a tempting target for symbolic behavior, including flouting the law. Now, the implications of it are that alcohol health warning labels or increased excise taxes or prohibitions or beverage alcohol advertising per se could even have a wrong impact. Is that your thesis? Or did I misread that?

Dr. ROOM. No, you read it right. I think you have got to be careful and look at it law by law. I do not think that there is a general rule for all laws. If you forbid alcohol to a segment of the population, notably to teenagers, then you offer a very attractive symbolic target, particularly as you are making a statement that if they can actually get some alcohol in their hands, then they have a claim on adulthood.

So in that particular arena there is a real paradox. We certainly have reduced the casualties for teenagers by raising the drinking age—there is no question about that—in our era. But I was raising the longer term question of whether we then are not laying up future trouble for ourselves, offering such a beautiful symbolic target for future generations. And we already may see some signs of that, I think, in some of the fraternity drinking episodes that have been happening on college campuses.

If you raise the taxes on something, though, that is not very symbolic. It does not offer such a symbolic target if when I go buy a can of beer it is no longer 50 cents, it is now 60 cents. And between those two extremes, it seems to me, it is important for legislators to keep attention to the possible symbolic meaning of the laws that they are passing.

Yes, the laws are a statement about the kind of society we want to have, but in these areas of expressive behavior, of behaviors like drug use and alcohol use that have so much symbolism attached to them, we can very easily offer a tempting target, and it varies with the particular legislation, I would argue, how much of the target it is offering.

Chairman GLENN. Dr. Vaillant.

Dr. VAILLANT. A simple way of monitoring whether your legislation is doing what you want it to or not is to watch how upset the beer, wine and whiskey lobbies are about what you are doing, because they are not dumb, and 50 percent of alcohol sold is consumed by problem drinkers and if you are upsetting that——

Chairman GLENN. Well, they get very upset. They are upset about these hearings. I am sure there are people here monitoring every word that is being said and reporting back to whatever the organization is in town today. I will not ask for a show of hands back here, but we already know about that because I know that some of my colleagues had calls from some of the industry groups over here inquiring about what my objective was, and they were asking me about this, and undoubtedly reported to some of these groups over in town.

So I am very much aware of the interest in this, but I also think it is incumbent on us to point out these problems and try and do something about them when we have 18 million Americans involved with this thing, or whatever the current figures are, and some of the tragedy that goes on, as I pointed out in my opening remarks very briefly. I think it is incumbent on us to try and see what we can do about it.

I would hope the industry representatives here can go back and say take the lead yourselves in trying to do something about this. Make sure that you cooperate, that you are not just out peddling everything without regard to how it is being used and what the tragic consequences of it are, and let's work together on some of these things.

I do not even know who I am talking to out there, but whoever it is, take that message back and then come back with a program and we will work with you on it. I will tell you that right now. I am not trying to take everything off the market or anything like that. But I am trying to do, in our little way here, whatever we can to try and address this problem that I think is so enormous. And while I am for all the hard drug problems, all the illegal drug—I am not for the problems, I am for the solution to those problems and what we can do about it.

But I also see this problem of the legal drug of alcohol being such a giant problem in itself. If you compare the two fairly, it almost makes one look small in comparison with the other one. So we have to concentrate on both of them.

Just one comment here, too. Dr. Room, you had another thing in your testimony I was interested in. You talked about the coordination between Federal agencies and the gaps in coverage. You referred to the fact that a high proportion of the Small Business Administration's minority business loans have been for liquor stores, and despite the problem and the high concentration of such stores in minority neighborhoods.

Now, that poses problems in several different directions. Obviously, we would like to say we are using our Small Business Administration loans for better purposes than that, but on the other hand, how do you say that when we are not outlawing liquor in our society—I am not proposing that here—that we then say, but we will not permit any loans to be made for the purposes of building a store to cater to that trade. We cannot do that, either. So it is a double-edged problem here I guess that we have.

Gentlemen, you have been very helpful. I appreciate your forthrightness here. We will get back to you, I am sure, with some additional questions that we want to include in the record. We appreciate your being here and we will keep in touch. I am sorry we have to move along to get our next panel on.

Thank you very much.

Our third panel this morning deals with biological markers for alcohol dependency and intolerance. The first witness is Dr. Henri Begleiter, State University of New York, Health Science Center in Brooklyn, New York; Dr. Shirley Hill, Western Psychiatric Institute and Clinic, University of Pittsburgh's School of Medicine, Pittsburgh, Pennsylvania; and Dr. Ting-Kai Li, Indiana University Medical Center, Indianapolis, Indiana.

We welcome you all here this morning. I have seen you back there in the room. You have been here all through the whole thing this morning, I believe. We are sorry we had to delay your testimony so long, but that was inevitable this morning, I guess.

Dr. Begleiter, would you lead off this morning.

TESTIMONY OF HENRI BEGLEITER, M.D., STATE UNIVERSITY OF NEW YORK, HEALTH SCIENCE CENTER, BROOKLYN, NY[1]

Dr. Begleiter. Thank you, Mr. Chairman.

First of all, Mr. Chairman, I would like to thank you for the opportunity of appearing before you and, indeed, we owe you a great deal of thanks for this opportunity, which is rather unique.

With your permission, Mr. Chairman, I would like to dispense with the reading of the testimony, which I have submitted to you and which is currently in your possession, and I would like to start off by providing you, if I may, a brief summary of some of the things which I have heard this morning and attempt to answer one of the questions which you raised just a moment ago concerning prevention.

Chairman Glenn. All right. That would be fine and your entire statement will be included in the record.

Dr. Begleiter. Thank you Mr. Chairman. And then proceed with a narrative description of the research which I summarized in my written testimony.

Chairman Glenn. Good. That is fine.

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[1] See p. 266 for Dr. Begleiter's prepared statement.

Dr. **Begleiter**. A couple of points I would like to make. First of all, as you have heard—and I think this needs to be emphasized—alcoholism is both a genetically heterogeneous disease as well as a clinically heterogeneous disease. That is to say, there are types of alcoholism for which genetics does not play a role at all and, of course, there are types of alcoholism for which genetics plays a rather important role.

In addition, there are different types of alcoholism with different clinical manifestations, and this is very important to bear in mind. This is no different from what we know in other areas of clinical medicine, such as there are different types of diabetes, there are different types of hypertension, there are different types of carcinoma. But I think it is very important to bear this in mind so as not to believe that alcoholism is alcoholism is alcoholism. It is a very heterogeneous disease.

Now, you asked before I think a very, very important question, namely, how might we get involved in some prevention initiatives? Is education worthwhile? And I think there is one answer which I would like to provide you with, which I have not heard yet, which is only a partial solution. And that is to address specifically the population which is the most vulnerable to develop to manifest the disease. So it is akin to, for instance, trying to address the entire black population about sickle cell anemia. Now, we do know that it is primarily a black disease. Nevertheless, we certainly cannot assume, and should not assume, that it is a disease which is present in all black families.

However, if we can in fact address those individuals which we know are the most vulnerable—and I do not mean to be addressing just those who are at risk. Risk is a statistical concept. Every child of an alcoholic may be considered at risk, but as you have heard before, not every child of an alcoholic is in fact going to become and alcoholic. And thus I would suggest that we need to develop markers, be they psychosocial markers or biological markers, which will help us identify not just who is at risk but who is vulnerable. And this vulnerability, if indeed one is looking at genetic vulnerability, is not in itself sufficient to bring about the disease.

So we are not talking about a nature versus nurture controversy here, but we talk about a gene/environment interaction. Again, this is rather similar to what may be found, for instance, in the area of cancer. One may have, for instance, an oncogene but that does not mean that one will go on to develop cancer. There are environmental variables which are absolutely necessary for the disease to manifest itself.

So I just wanted to provide you with those few thoughts before I go on and tell you some of the research with high risk populations.

Some 25 years ago I was instrumental in the development of a technique called evoked brain potentials which has now found its way into the chemical arena, primarily in the field of neurology, which allows us to record in a non-invasive, innocuous fashion, the electrical activity of the brain to various challenges, to various stimuli. And so with this technique one can relatively easily look at the dynamic pattern analysis, the dynamic properties of different systems in the brain on a millisecond to millisecond basis, and better able to understand how the brain processes information.

This can be done at various levels, so one can assess the integrity of various sensory systems such as one may want to assess, for instance, how an auditory stimulus is processed, starting all the way at the ear, all the way up to the cortex, which is the highest level in the humane brain. One can begin to follow, if you will, that stimulus all the way up to the cortex.

One can do the same thing for all sensory areas of the brain. But more importantly, and more exciting, one can also get the subject, the individual, involved in various cognitive tasks which allows us to look at how hard the brain is processing rather complex information.

What one derives is a pattern of activity which can be analyzed mathematically and which can be very, very helpful in understanding the information processing in the normal brain as well as the abnormal brain.

So in possession of this technique which, as I say, we were somewhat instrumental in developing some 25 years ago, we have for the past 25 years studied chronic alcoholics. We have over the years observed a number of deficits—that is neurophysiologic deficits, specific brain deficits—at various levels of function. That is, some of these deficits are sensory, involve vision, involve audition, involve the somatosensory system as well as more complex deficits which involve the higher integrator centers of the brain.

We discovered that there were numerous deficits, and all these deficits have, needless to say, been observed by a number of other people in various laboratories throughout the country and, indeed, throughout the world.

And we asked the question, approximately 10 years ago, are these deficits in any way reversible? Is it possible that these so-called brain deficits are temporary, that is to say reverse, with total abstinence? And so we began to look at alcoholics who had been abstinent anywhere from 4 months to 5 years, and found, surprisingly so, that many of the deficits which we had initially observed and reported, indeed recovered over time—and this was very encouraging—but, for a handful of deficits which had not changed at all over time, remained and in fact were to be found in chronic alcoholics abstinent for as long as 5 years.

So we were puzzled because initially we had hypothesized, we had postulated, that all of these deficits represented the natural consequence of alcohol abuse. Alcohol is a relatively neurotoxic agent, so we invariably assumed that if you abuse alcohol it will in fact bring about a great deal of damage to the central nervous system. But we could not account for the fact that some of these deficits reversed and others did not change at all, even after 5 years of abstinence.

Around 1980, with the advent of population genetic studies such as those described by Dr. Goodwin, initially conducted by him and his colleagues in Denmark, we became aware that sons of alcoholics were at very high risk for developing alcoholism. So we began to entertain the possibility that some of the deficits which did not reverse at all in chronic alcoholics abstinent for 5 years possibly did not represent a consequence of alcohol abuse but anteceded the development of alcoholism.

To that end we initiated a series of studies with high risk populations which were initiated in 1980 and are still going on today in our laboratory, as well as those of many other laboratories, including Dr. Hill who is sitting here on my left. In 1984 we published the initial findings which reported that young sons of alcoholic fathers, between the ages of 6 and 13, who indeed had never had any experience whatsoever with either alcohol or any illicit drug, manifested neurophysiological deficits, CNS brain deficits, which were identical to those which we had initially seen in the fathers and which did not reverse.

Chairman GLENN. Let me stop you there just a second. In those people then, if they became alcoholic and then got off alcohol, did the deficits correct themselves with those people, like you said it did with some others?

Dr. BEGLEITER. No.

Chairman GLENN. Those people who had alcoholic parents, their deficits—they had the deficits to begin with. Then they became alcoholic. If they then went on the wagon and did not have any alcohol, the deficits would not correct themselves, as you said happened in some cases before? Is that correct?

Dr. BEGLEITER. Those studies were not done. Those studies, Mr. Chairman, require prospective longitudinal studies, which are very expensive and which you have heard much about throughout these hearings. So that the only thing we have done is to identify a set of neurophysiological deficits in young sons of alcoholic fathers. These sons need to be followed up. Some of them will become alcoholics, and in order to ask the question which you are asking, which I think is a very pressing and important question, we need to conduct longitudinal studies, which are very time consuming, labor intensive and expensive.

Chairman GLENN. Are the deficits treatable in advance? I guess that is the threshold question.

Dr. BEGLEITER. Okay. And I do not know the answer to that either, Mr. Chairman, regretfully so.

What is important and scientifically exciting is that the deficits in these high risk boys are very similar, if not identical, to the deficits you see in abstinent fathers. Again, this is not only in our own laboratory but the results observed in several other laboratories.

Moreover, I think what is important to mention is that if you look at the general population of alcoholics without any due regard for possible familial transmission—that is, for any kind of family history of alcoholism—if you just take a sample from a treatment facility of alcoholics without asking any questions about family history of alcoholism, the prevalence of the deficits in the offspring is approximately 35 percent.

If, on the other hand, you begin to look at a rather different sample of alcoholics and their offspring, namely alcoholics where there is a family history of alcoholism, then the deficit is to be found in at least 70 percent.

Chairman GLENN. The deficit, is it racial or ethnic in nature?

Dr. BEGLEITER. I cannot answer that because we do not have sufficient data to answer that. In our case, for instance, we looked primarily at minority individuals, primarily black and Hispanics, with a small percentage of white.

Chairman **Glenn**. I was thinking in particular—I do not know whether the reputation is deserved or not—but the reputation and the conventional wisdom is supposed to be that American Indians have a very low tolerance for alcohol. I do not know whether that comes from too many western movies we have watched or what, but that is the reputation. And I have read about the one town where almost everyone in the town on one of the reservations had problems with alcohol.

Now, have you run any of these deficiency studies for that particular group?

Dr. **Begleiter**. We have not, Mr. Chairman. And the problems of Indians and Asians I think will be discussed somewhat later by Dr. Li, I believe.

Chairman **Glenn**. Go ahead. I did not mean to interrupt.

Dr. **Begleiter**. No, that is quite all right. We have no data. We currently have no data on Indians, nor have we a sufficiently large sample to examine the question which you raise, namely, the issue of ethnicity. So I really cannot answer your question, Mr. Chairman.

It is an interesting question and one which is being pursued but requires, of course, rather difficult sampling techniques in order to equate for a number of other variables. But I think what is important is to realize that here we have a potential marker. That is to say, we are using a measure. These brain events are highly heritable events. The rate of heritability is very high for these brain events.

So we are looking at something which we know to be heritable. And with this in mind, we are able to identify in a rather large proportion of a specific high risk population a rather specific abnormality or set of abnormalities which are similar in young sons as well as the fathers.

And so we feel that this is most encouraging and it has two immediate implications. One, to go back to the issue of prevention, we feel that it may be worthwhile to address some of our energies and efforts toward a population where you know there are some abnormalities, be the biological or psychosocial, but when you do know that there seems to be at least a somewhat higher degree of vulnerability.

Second, these particular deficits are of great help to us in helping us better understand the etiology, if you will, the causes of the disease, again, in a specific segment of the alcoholic population. And I certainly do not mean to imply that by looking at high risk individuals we will understand all of alcoholism. We will not, but it will help us certainly better understand what we believe to be a population at very high risk for the disease.

Chairman **Glenn**. Thank you very much.

Dr. Hill.

TESTIMONY OF SHIRLEY Y. HILL, Ph.D., WESTERN PSYCHIATRIC INSTITUTE AND CLINIC, UNIVERSITY OF PITTSBURGH SCHOOL OF MEDICINE, PITTSBURGH, PA[1]

Dr. **Hill**. Thank you, Mr. Chairman.

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[1] See p. 287 for Dr. Hill's prepared statement.

If I may, I would like to depart from the written comments which I submitted, also, and make some general comments in response to questions that were raised this morning that I think are very important.

Chairman GLENN. Fine.

Dr. HILL. I would like to reiterate what Dr. Begleiter said regarding prevention. While I think it is very important that we try to prevent problems for the population as a whole, I think it is equally important, if not more so, to target some of the groups who are at particularly high risk for developing alcoholism.

One of the payoffs from doing the kind of research that Dr. Begleiter and I are doing is that at some point in time we may be able to identify people who are at risk through use of these markers.

The second point that I wanted to make about markers is that once we have identified a marker, we may not be able to necessarily reverse the course through changing the marker itself.

Chairman GLENN. Let me interrupt just a moment here. When we are talking about markers—and I will show my medical ignorance here—are we talking about a chemical deficiency? Or are we talking about a neurological path that never was developed like others? Are we talking about something that can be chemically treated or medically treated? Or is it a genetic DNA thing that we do not know how to treat yet? Or all of the above? What is it?

Senator SASSER. I am glad you asked that question, Mr. Chairman, because I do not know what a marker is, either.

Dr. HILL. That is a very good question. A marker is something that simply points us to who is at risk. In other words, if you did a study and you found that all people who had blonde hair were at higher risk for developing coronary artery disease, that might enable you to put certain people into prevention programs. The blonde hair may have no functional significance in the same way that the brain wave characteristics that have been described may or may not ultimately be something that one could change, and therefore use to prevent alcoholism.

Right now we are simply saying that having this marker would enable us to target certain individuals as being at higher risk.

Chairman GLENN. Some of these markers might be chemical, some might be EKG, some might be your blonde hair you talked about?

Dr. HILL. Exactly.

Chairman GLENN. It is just an indication that a greater number of people with that characteristic seem to develop a certain problem?

Dr. HILL. That is right.

Chairman GLENN. Okay.

Dr. HILL. In some cases the marker may have something to do with the etiology of the disorder and in that case it would be even more helpful—identifying the high risk individual and moreover explaining how he/she got that way.

Chairman GLENN. But these are, I presume—and I do not want to interrupt your testimony here until you get finished, but I will anyway for just a moment here—these are not things that would have been changed by alcoholism? In other words, a person is an alcoholic, then you measure them. There might have been some changes because of the alcoholism itself.

Dr. HILL. That is right.

Chairman GLENN. That would be a marker that they are an alcoholic but it was induced by the alcoholism, if you follow that convoluted statement.

Dr. HILL. That is right. Traditionally, we have looked for markers among alcoholic individuals, but with time researchers became more sophisticated and said, let's start looking at relatives of alcoholics who are not themselves alcoholics so that we can see if they vary from a normal control group, and then maybe we will be on to something.

So, yes, we are now at the point where we have identified the markers that truly are risk markers and not what I like to call episode markers. They are simply markers that reflect an episode of drinking or alcoholism.

Well, I would like to now describe our own research effort. Approximately 5 years ago we were funded by NIAAA to begin to look at neurobehavioral markers and blood markers for risk for alcoholism. To do this we thought that it would be best to identify families where there is heavy genetic loading.

You have heard today that there are at least two types of alcoholism; one that appears to be familial and one that appears to be less familial. Our goal was to identify not only the familial form but one which was exceedingly likely to produce alcoholism in successive generations.

So to do this we identified alcoholic families where there was a minimum of two alcoholic male siblings, and in doing this we identified families who were very heavily loaded for alcoholism, such that almost 40 percent of the first degree relatives of these alcoholics were alcoholics themselves.

Now, just by way of note, a first degree relative is a father, a mother, a child, someone that is only one generation removed from the individual. There was, of course, increased alcoholism among the second degree relatives and third degree relatives as well.

We enlisted the cooperation of these families and then began to look for the markers for risk. I am pleased to tell you that that research has already paid off in that we have now found evidence for two neurobehavioral markers as well as a blood marker that has identified high risk individuals.

First, the neurobehavioral markers. We have identified in our brain wave studies—which are similar to Dr. Begleiter's—we have identified children of alcoholics who are showing a reduction in amplitude of a particular brain wave, the same wave that he is studying. We also find that the siblings of these alcoholics, who are not themselves alcoholics, also show changes. So we think that this is a robust change that says to us that we can pick out individuals who are at higher risk.

Now, it may turn out ultimately that this brain wave may have some significance. It may be telling us that one of the things that is transmitted from generation to generation is a tendency for people who are going to become alcoholic to process information in a different way than people who are not. It is probably too early to tell exactly what that marker may mean. But at this stage of the game I think that there are enough laboratories that have found this marker that we can tell you today that we do have at least one neurobehavioral marker that we can agree on.

The second neurobehavioral marker that I have been particularly impressed with is one having to do with static ataxia, or sway. I think you heard earlier that Dr. Lex is studying sway in individuals who are given alcohol, finding that those who are at higher risk for developing alcoholism have less sway.

Well, our strategy was to study sway in children without giving them alcohol and simply see if there was any difference between children of alcoholics and nonalcoholics. We were following the lead of other laboratories that had accidentally come upon the finding that children of alcoholics swayed more when asked to stand steadily in one position at rest and to not move. We were somewhat surprised to be able to replicate this finding, because we could not offhand think of any reason why children of alcoholics should necessarily sway more at rest than children of nonalcoholics. And yet we now have two studies in our laboratory, and other people have found similar results, that suggest that this too is a neurobehavioral marker that may pay off.

I might also add that because we have a three-generation sample, heavily loaded for alcoholism, our goal is to then follow these children for 10, maybe 20, years and see if our markers pay off. This, as many people have pointed out, is a very time consuming task but I think that we will be able to find out if some of these markers pay off in less than 20 years because many of the children in this third generation are in their upper teens, so that soon they will be entering the age of risk.

The third marker that I wanted to talk about is our genetic linkage finding. Every subject who comes into our laboratory from this three-generation study goes through a variety of tests, including being asked to give a blood sample. So from all the data that we have collected to date we have been able to determine that there is genetic linkage between risk for alcoholism within a family and the presence of a variant of a blood marker.

Now, I should hasten to add that this does not mean that we have found a gene for alcoholism. I wish I could tell you that we have. But I think we have come closer in that by sampling markers throughout the human genome, some on chromosome 1, some on 9, and so on, we have now been able to narrow our search so that we think that we have found evidence for a gene on chromosome 4. Further study may prove us wrong, but I think the value of this finding is that other research groups will begin to look for this gene, which I think is very exciting.

You may be familiar with the story with the attempt to find a genetic basis for depression. There was an announcement a few years ago that someone had found a genetic linkage between a blood marker and depression in an extended Amish family and interestingly, right after that happened there were a number of other research groups that then became interested and pursued this finding so that now I think that we have some evidence that there is a genetic basis for depression. So I am hoping that our finding will generate more interest in searching for an alcoholism susceptibility gene.

So where does this lead us in terms of prevention? My hope would be that at some point in time, if a family came to us and said, there is a single person in my family who is alcoholic, or there are two, or there are three; I am concerned about my children; can you give me any idea about the risk? That we might be able to administer a battery of tests and give them a better notion of what the risk would be should they or their children drink.

I would be very opposed to, obviously, screening the entire population for risk through such means. But I think everyone deserves the right to know what their risk is if they can find from scientists what that is.

So, in conclusion, I think that our group as well as others have found a number of exciting markers that will ultimately be of some benefit in guiding persons at high risk into prevention programs.

Thank you.

Chairman GLENN. Thank you, Dr. Hill.

Dr. Li, you have done a lot of animal studies, I understand. We welcome your testimony.

TESTIMONY OF TING-KAI LI, M.D., INDIANA UNIVERSITY MEDICAL CENTER, INDIANAPOLIS, IN [1]

Dr. LI. Thank you very much.

Before I start I would like to make a couple of general comments as well concerning points that have been brought up by others.

One has to do with the prevention efforts, and I think it is important that we look at prevention with a view of what we are preventing. It is very important to first of all target the goal of the prevention. Are we preventing fetal alcohol syndrome? If so, then I think we need to target women of childbearing age, for example, rather than men as a primary group.

And second, for instance, if we are trying to prevent driving accidents, then we should be targeting another specific group and adopting different measures, one of which has been alluded to already, namely, making driving more restrictive on those who drink.

So I think it is very important then that we target the population, and I think Dr. Begleiter and Dr. Hill have already addressed the issue of the high risk populations that are children of alcoholics and the importance then of being able to identify within that group those who are vulnerable. And that is what the marker studies are all about. A marker, in another way of calling it, is a diagnostic test, and the values of diagnostic tests are one, sensitivity, and the other specificity.

Now, if we have a marker that pertains to the children of alcoholics as a group, then we have a sensitive test because we have covered everybody. But, to be specific, to be good, it has to be both that and specific, which means that within the population of children of alcoholics that you are able to identify those who are specifically at risk.

For example, the marker that Dr. Hill talks about, perhaps may be 40 percent specific, and by developing a constellation of tests, then you can make it 95 percent specific. This constellation of tests then would be a good approach to finding those who are vulnerable.

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[1] See p. 324 for Dr. Li's prepared statement.

Now, let me go on to telling you what our interests are in our laboratory. We are interested in knowing why people drink and why some people drink too much. Now, alcohol drinking is a quantitative trait. Quantitative traits like body weight and height are affected by multiple factors, multiple genetic factors as well as multiple environmental factors.

So there would not be any one gene that affects this trait, but there will be many, among which some may be more important than others. Our interest is in looking at the genetic and the biological factors that influence drinking behavior in humans and in experimental animals. Our studies in humans are more on the biochemical and molecular genetics level, and the animal studies are in animal models. There are different reasons for this, and I will go into these.

Now, when we look at biological responses to alcohol that can serve potentially as feedback loops to influence drinking behavior, we find that a number of them—alcohol drinking behavior, as well—are influenced by genetic factors. For example, the frequency and quantitative aspects of drinking is under genetic control. We know that to be the case in humans.

We know in humans that the brain sensitivity to alcohol varies quite a bit and that is influenced by genetic factors. And we know that alcohol metabolic rate, and maybe even the incidence of alcohol-induced liver disease and brain damage are also influenced by genetic factors.

Recent studies in humans have now shown that genetic differences in the alcohol elimination rate can have a profound influence on drinking behavior and the development of alcoholism. Drinking elimination rate can vary as much as 3-fold among humans. The difference between individuals may be as large as 3-fold. We think a large part of that is due to differences in the enzymes that metabolize alcohol. The two enzymes that are primarily involved are alcohol dehydrogenase and aldehyde dehydrogenase.

The first enzyme takes alcohol to acetaldehyde, and then the second enzyme takes it from acetaldehyde to acetate and from there it is converted to water and carbon dioxide. The interesting thing is that in humans both of these enzymes show genetic polymorphism, meaning that there are genetic variants of these enzymes that we can now detect. Our laboratory has isolated all of these forms and shown that they are different in catalytic properties.

Chairman GLENN. Have we taken these to ethnic differences and racial differences yet?

Dr. LI. Yes, we have.

Chairman GLENN. Because I have a question here, and I will just get it in right now, about what I think is called the "alcohol flush reaction." There was a report on NPR, National Public Radio, yesterday morning about problem drinking in Japan that suggested that people with this particular reaction to alcohol were able to condition themselves to tolerate massive quantities of alcohol.

Now, are there any endogenous or environmental factors you are aware of that would protect one against becoming a problem drinker?

Dr. Li. Yes. I will be addressing this.

Senator Glenn. All right. Good.

Dr. Li. So, there are genetic differences in both of these enzymes, and the alcohol dehydrogenase polymorphism shows up in orientals versus Caucasians versus blacks. Now, you have asked about the American Indians. We have studied several different American Indian tribes, mostly from the Southwest, and they look more like Caucasian than oriental.

I will now go on to the alcohol flush reaction. The alcohol flush reaction occurs in about 50 percent of orientals. The degree of flush varies. The intensity of flush varies. But we know from studies in the Japanese and Chinese that there is a form of alcohol dehydrogenase present in about 95 percent of orientals that is different from whites.

As far as aldehyde dehydrogenase is concerned, 50 percent of orientals lack a certain aldehyde dehydrogenase form. This is the so-called mitochondrial low-Km form, and this is the form that is primarily responsible for eliminating acetaldehyde.

Now, it is important to eliminate acetaldehyde because it is very much more toxic than alcohol and when it is even in very low concentrations can cause release of vasoactive peptides and other substances, and that is positively correlated with the alcohol flush reaction. So it is the genetic deficiency of this enzyme that is probably a necessary condition for the alcohol flush.

The variation and degree of severity of the flush may be conditioned by whether you are homozygous or heterozygous for this deficiency in aldehyde dehydrogenase and also depending on what your enzyme pattern is for alcohol dehydrogenase, because that is the enzyme that produces acetaldehyde.

Now, how does this relate to drinking behavior? Well, studies in Japan have shown that people who flush will drink less and drink less frequently. Also, whereas the incidence of the deficiency of the aldehyde dehydrogenase may be 50 percent in the general population, in groups of alcoholics, the incidence of the deficiency of this enzyme is only 2 to 5 percent. So, therefore, the flush reaction is a protective mechanism against heavy drinking and it is negatively correlated with alcoholism.

What use has this in terms of therapy? Well, actually a therapy has been developed even before we knew about the alcohol flush reaction and that is the use of Antabuse. How Antabuse works is exactly through this mechanism. It inhibits aldehyde dehydrogenase and people who drink while they are on Antabuse develop this flush reaction. Well, you might think that this should be very effective. Studies have shown that the efficacy varies and, of course, you would expect this because the problem is one of compliance. The alcoholic has to want to take that drug first, and if they do not take it, they do not have the reaction and obviously then they are not protected against drinking.

Let me then go on to the second area about animal models, and that may have some relevance to children of alcoholics. There are certain things we can study in humans and certain things that are not easily done. We are interested in whether the phenomena of tolerance or brain sensitivity is related to alcohol drinking, and this is not that easy to do or manipulate experimentally in humans, so we go to animal models.

Our approach is to try and develop through genetic means animals that like to drink alcohol. This is voluntarily since they have food and water available at all times. Then you give them alcohol; that is 10 percent alcohol, or even as high as 30 percent alcohol, which would be 20 and 60 proof, respectively, unflavored, and let them choose between the water and the food and the alcohol and these animals will choose to take lots of alcohol.

We do this through genetic means by selecting from different populations animals that like to drink and animals that do not like to drink. We then mate the ones that like to drink, and through multiple generations we develop a line that breeds true for alcohol preference. By breeding together the other animals that do not like to drink we have developed an animal line that shows the opposite behavior.

This per se is proof that voluntary alcohol drinking behavior is under genetic control. But what is more important about developing these kind of models is that now you can look at their response to alcohol and see how they differ, the two lines, and that may teach us something that could be useful in treatment and prevention strategies.

I ought to say that the preferring line essentially satisfy all the pharmacological criteria for an animal model of alcoholism, which is that, first of all, they drink lots of alcohol, their blood alcohol concentrations are high, into the intoxicating range; in long term drinking, they develop tolerance, they develop dependence, and more importantly, they find alcohol rewarding. In other words, they will work in order to get the alcohol. And that is one of the tests that we use for addictive drugs. Drugs are shown to be addictive in humans and in animal models with techniques that show that they will work in order to get the drug, for example, through bar pressing. The preferring animals will work to get the alcohol, but nonpreferring animals will not.

Several intriguing findings have come forth. Number one is that the preferring animals find alcohol reinforcing at the low blood alcohol concentration like 50 milligrams percent, or two drinks for a human being. They find that rewarding, whereas the non-preferring animals are not affected.

Second, the preferring animals develop tolerance to the behaviorally impairing effects of alcohol at higher doses more quickly and they keep it for a very long time as compared to the non-preferring animals.

Now, if you look at children of alcoholics, from what Dr. Lex and Dr. Hill have said, their insensitivity to the intoxicating effects of alcohol, both behaviorally and with psychometric testing, may be an expression of tolerance, for example. So this is a hypothesis that can certainly be tested.

Another finding that you have already heard mention of, is that serotonin is a possible mediator. The preferring animals exhibit lower levels of serotonin in certain brain areas, specifically in the areas of the brain that are thought to be important in brain reward recognition. This lowering of serotonin is quite a robust phenomenon because now we have started a second experiment of selected breeding and we find the same thing.

So what does this mean? Well, we can actually test drugs to see whether they will change drinking behavior, and indeed we can do this with a drug that raises serotonin in the brain, and this is the example you have already been given—one of them is fluoxetine, now marketed by Lilly as Prozac. We find this to be effective in reducing drinking. Moreover, we can even become more specific and look at so-called serotonin receptor sub-types and find effective drugs that are more specific to certain kinds of serotonin receptors than others.

So this is, then, the kinds of research that we can do with the hope that we can develop drugs that will modulate craving, and this is something that, again, Dr. Goodwin has alluded to.

Now, in terms of final recommendations, I think the work in terms of discovery of enhanced tolerance and also of differences in enzyme metabolism are important to look at as, "markers" of susceptibility. In this regard, I think it is important that we now can determine the genetic differences in the enzymes of alcohol and aldehyde metabolism by using blood tests as well. We no longer are dependent on doing liver biopsies. We have recently been able to do genotyping by using white blood cell DNA and type all the alcohol dehydrogenase polymorphisms as well as the aldehyde dehydrogenase polymorphisms. So these are some of the future directions that could be useful in terms of treatment and prevention strategy.

Chairman GLENN. Thank you. It was very interesting, all of you.

Do the biological markers change over time? Change with environment? Would you biological markers change possibly if you moved from New York to California, or vice versa?

Dr. BEGLEITER. We have followed some of these kids for a period of 4 years, testing them every year, and we find that the markers are remarkably stable.

Dr. HILL. I would like to add, too, that with the blood markers we are looking at something that is an inherited characteristic of the individual, something as simple as the ABO system, the system that is used when you have to have a transfusion, and that does not change with environment.

Chairman GLENN. Your blood chemistry undergoes a temporary change when you go to high altitude or low altitude. If you go to Vail to ski or something, you are at 8,000 feet. You have a couple of days and things are not quite right until you get adapted a little bit and there is an actual blood chemistry change occurs. Does that change your markers?

Dr. HILL. The markers I am speaking of are genetic blood markers so that they—I mean, you have Type A blood from the time you are born until you die, and that is the kind of blood markers that I am studying.

Chairman GLENN. Have you noted any difference, Dr. Begleiter, in altitude or changes across country or anything?

Dr. BEGLEITER. We have not done that, Mr. Chairman.

Dr. LI. I might add that the alcohol aldehyde dehydrogenase content in liver does change with liver disease or with drinking, but what we are looking at are the actual genes for these enzymes, so we are looking at genetic material directly.

Chairman GLENN. Dr. Hill, the neurobehavioral or electro-physiological differences that you have noted in children, are these the same for both male and female children? Is there any difference?

Dr. HILL. We have looked at approximately equal numbers of girls and boys to date and we find the same findings for both boys and girls. I should add, though, that our study is designed around male alcoholics that we get from treatment centers, and their families. We are hoping to embark on another study where we select families where there is a pair of women who are alcoholic and see if their children differ from children from families of male alcoholics.

Chairman GLENN. How about differences in non-Caucasian children; have you gotten into that yet?

Dr. HILL. I cannot answer that but I believe Dr. Begleiter's children are non-Caucasian.

Chairman GLENN. You have done some different ethnic group studies?

Dr. BEGLEITER. We have looked at primarily non-caucasians and our findings are identical to our Caucasian population in California and the one that Dr. Hill is studying in Pittsburgh.

Dr. HILL. Right.

Chairman GLENN. We are concerned, obviously, with programs here that are going to not only screen and treat but are going to prevent alcoholism and while you are dealing with very specific parts of this overall problem, I would appreciate any comments just in closing that any of you might make.

Is there such a thing as a model anti-alcohol abuse program that you would like to see enacted? Or is there a Federal role that we should play in this that is not being played now? Or do you just need more money for research with existing programs? I can give an automatic yes to that on behalf of you probably before we—I will take care of that question myself.

Do you have any suggestions on prevention, other than the education approach that Dr. Bowen talked about yesterday? Why don't we just go across the line here.

Dr. Begleiter?

Dr. BEGLEITER. Well, let me reiterate what I said before. I think the best prevention initiatives, the most rational ones, are always the ones which target a very specific population as opposed to general prevention initiatives where typically the efforts are diluted so that if you are addressing a child about the possible deleterious effects of drinking and this child has never seen an alcoholic, does not have any alcoholics in the family, it is very difficult for that particular child to understand that message, be it positive or negative. And the message is received quite differently if indeed you are addressing the child of an alcoholic who has extensive experience with alcoholism on a daily basis.

So I think it is absolutely critical to identify very specific target populations and then to begin to assess essentially the efficacy of your prevention initiative as opposed to initiating a large, broad prevention initiative which you know is not likely to be very successful.

Chairman GLENN. Dr. Hill?

Dr. HILL. I would like to reiterate that I think educational efforts should be targeted at the high risk individual because in talking to these families I have learned that many people who had alcoholic parents and are themselves alcoholic had made no connection between the parent's alcoholism and their increased risk. This comes somewhat as a surprise to them.

So my hope would be that by providing genetic counseling to these high risk individuals that we could provide them with scientific data that might be more believable to them than just knowing that they have a family history.

Chairman GLENN. Maybe that awareness, though, is changing somewhat over the past few years. In fact, just the last couple of years the little booklet, "Adult Children of Alcoholics" went into this in considerable detail and was on the New York Times best seller list, I think, for what was it? Like 7 or 8 months. I think it headed the list. And so I think some of the awareness is getting out and perhaps our hearings can perform a tiny little bit in addition to that also.

Dr. Li?

Dr. LI. Well, being a clinician, I used to do alcoholism treatment. I do not anymore. I do all research. The only thing I can say is that I think the treatments we have now are purely empirical and it is based on what works, or what may work or what may not work, and it is polypharmacy. We use whatever approaches we can have our hands on because we really do not have any understanding. The understanding has to come from research.

But even with empirical approaches, outcome evaluation I think is the most important thing to have, along with any kind of treatment or prevention program that you initiate. And the importance is to have an outcome evaluation component so that you know from solid data whether what you are doing is effective or not.

Chairman GLENN. Thank you all very much. We may wish to have additional questions submitted. We hope you would respond to those questions so they could be included in the record. Our one problem we always have with hearings like this is we want a broad spectrum of people coming in but then you come in with the work you are doing and the testimony you give is so interesting we run over in time, and so we do appreciate your forbearance with us this morning and sticking around here.

It was a long hearing this morning, but we do appreciate it very much and we will be keeping in touch with you and on behalf of all of us we wish you very good luck in your research. It is on behalf of all of us.

Thank you very much. The hearing will stand in recess subject to the call of the Chair.

[Whereupon, at 12:25 p.m., the Committee adjourned subject to the call of the Chair.]

APPENDIX

PREPARED STATEMENT OF OTIS R. BOWEN, M.D.

Chairman Glenn, and Members of the Committee, I appreciate the opportunity to testify this morning to discuss the problems of alcohol abuse and alcoholism in our Nation today. It is a pleasure to unveil before the Congress, for the first time, my initiative on alcohol abuse and alcoholism.

With me today is Thomas Burke, Chief of Staff of the Department of Health and Human Services; Robert Trachtenberg, Deputy Administrator of the Alcohol, Drug Abuse, and Mental Health Administration; and Dr. Enoch Gordis, Director of the National Institute on Alcohol Abuse and Alcoholism.

Dr. Gordis will describe our alcohol research programs for you in greater detail later in this hearing. Tom Burke will elaborate on our progress with my alcohol initiatives.

But, first, I want to communicate to you my own deep concern about the tragic and needless toll taken by alcohol abuse and alcoholism on our society and our people. And then I'd like to outline for you the major initiative I have undertaken.

Effects of Alcohol Abuse and Alcoholism

As a physician back in Indiana, I saw first hand many of the medical problems surrounding alcohol abuse and alcoholism and witnessed the suffering caused to both patients and their families.

There's much we already know about the devastating and often hidden effects of alcohol abuse and alcoholism on our society.

Alcohol abuse and alcoholism harm families and contribute to a wide range of other health and social problems -- such as birth defects, accidents, mental illness, other drug use, family violence, teen suicide and homelessness.

Alcoholism and alcohol related problems are estimated to cost the country more than $117 billion dollars a year in everything from health care bills to loss of productivity. Alcohol costs our society plenty -- and much of this comes out of the public treasury, in one way or another.

If we could tackle successfully the alcohol problem in society today, many of the other problems we face would be dealt a considerable blow -- if not eradicated entirely.

In January 1987, I submitted the Sixth Special Report to the U.S. Congress on Alcohol and Health. That report contains ample evidence that our understanding of alcohol-related problems continues to increase and that this growing body of knowledge has the potential to alter significantly the way we prevent and treat alcoholism and alcohol related problems in the future.

Development and Goals of National Initiative

Early last year, I set out to build on what's already been done in the alcohol abuse and alcoholism fields and to see if, working together with health professionals and other interested people outside the field, both in the public and private sectors, we could make the problems of alcohol abuse and the disease of alcoholism highly visible and an urgent priority for the American public. It was reported to me that both the media and the American public were tired of the alcoholism issue. Studies have shown that the number of feature and news articles and story lines on alcohol related problems in radio, television, and the print media were all significantly diminished from what they had been -- while at the same time, the toll on our nation in terms of lives wasted, health care costs, and productivity lost had never been higher.

I became convinced that the high visibility of my office could be used to create a national agenda to prevent and reduce alcohol related problems. This is one of my priorities as Secretary. I believe we're making good progress.

One year ago, I convened a distinguished panel of experts in alcohol abuse and alcoholism prevention, research, education and treatment to advise me on what was currently being done and what I, as Secretary, could do to stimulate further action.

I asked for and received from this panel -- after several months of hard work -- a frank and unedited report which detailed their findings in five major areas of study: Research and Practice; Education, Information and Media; Treatment; Public Policy and Legislation; and Federal Leadership.

Working from that exhaustive report, I decided on a comprehensive national initiative which I announced at our first-ever Secretary's National Conference on Alcohol Abuse and Alcoholism last November.

I believed then, and believe even more so today, that we need to develop a public-private partnership committed to raising public awareness. Alcohol and other related alcohol problems are robbing us of many of our national treasures - including our youth who are our hope for the future.

My National Initiative on Alcohol Abuse and Alcoholism is a multi-pronged effort. It includes components for:

- better coordination of HHS alcohol-related activities;
- speedy transfer of research findings to the clinical practitioner;
- fostering research into prevention efforts;
- improving health professional education;
- mobilizing Federal efforts to encourage small businesses to

participate in employee assistance programs;

- a public/private sector national media and information campaign;
- targeting new efforts aimed at identified high-risk populations, such as women, specific ethnic groups, and children of alcoholics; and
- establishment of a privately-run National Citizen's Commission.

The Role of Health Care Providers

In trying to design a comprehensive strategy to combat the alcohol abuse and alcoholism problem, we recognized two areas in which progress could be made to help health care providers intervene -- identifying the problem and barriers to treatment.

We hear of under reporting by physicians of alcoholism and alcohol abuse as contributing causes of death. There are many reasons for this, but it in great part stems from a concern for the feelings for the family left behind. I was and still am troubled when I hear reports of the tendency on the part of some of my colleagues in the medical community to overlook a patient's alcohol problem when seeing the patient for other reasons, some of which probably are alcohol related.

It's usually a primary care provider who makes the initial diagnosis for all of a patient's health problems, but this hasn't been the case with alcohol-related problems.

In a 1982 study, 70 of 271 problem drinkers were found to have been seeing a physician for various reasons at the time the patients first noticed their drinking problem. Only 45% of those seen, however, were asked by their physician about their drinking, and only one-fourth of these were either encouraged to reduce their intake or warned about the health hazards of drinking. And just 3% of those heavy drinkers were referred to treatment by their physician -- 3%!

I think members of the medical community have a unique opportunity to educate their patients about alcohol abuse. But one 1986 study pointed out that primary care physicians have had little success in diagnosing or treating alcohol abuse, other drug use, or mental disorders.

A number of factors may contribute to this difficulty.

For one, patients themselves may resist, either because they haven't admitted their problem or because of the stigma attached to both alcohol abuse and alcoholism. Physicians may sympathize with them and be reluctant to force the issue or be fearful of losing their patients.

But another reason may be because training in alcoholism diagnosis, intervention and treatment is only a very limited part of most health professions education programs, medical schools and residency programs included. And as a result, graduate medical professionals have not established an approach to the problem and do not feel confident discussing the problem with patients.

One major force mitigating against such curricula, at least in medical schools, is probably the structure of academic medicine itself. Because it's based on the acute care medical approach, it's not really designed for chronic diseases like alcoholism or other addictions or other behavioral problems.

Another reason alcohol-related curricula may be inadequate to reflect the extent of the problems we face in this area may be because faculty members themselves have not been prepared to teach about it - and perhaps more importantly, it is not emphasized on board examinations, including examinations given by specialty boards.

Most experts say there also aren't as many continuing medical education programs on alcohol abuse and alcoholism as there should be.

Of physicians polled by the Journal of the American Medical

Association in 1982, 71% said they felt ill-prepared for, or ambivalent about, making alcohol diagnoses on patients.

Given that primary care providers often see alcoholics and alcohol abusers before alcohol treatment specialists do, they have got to be alert to every sign of a problem -- especially among our young people, many of whom may be alcohol abusers, may have a predisposition to alcoholism or may be the children of an alcoholic parent or parents. Research into the special problems of children and families of alcoholics has increased dramatically in recent years, and it is imperative that our health professionals pay special attention to them in their work. They need to be up to date in the knowledge they have about alcohol and alcohol related problems.

My alcohol initiative includes a recognition of these problems of medical education and intervention to help alcohol abusers and alcoholics -- and we've taken several actions I think will help.

Our Health Resources and Services Administration and our Office of Substance Abuse Prevention are now funding a project to improve the ability of primary care providers located in community health centers and maternal and child health programs to diagnose, refer, and treat alcohol related medical problems and alcoholism.

Our Alcohol, Drug Abuse, and Mental Health Administration has initiated a program which focuses on primary care providers through the development of curriculum models for alcohol and other drug abuse teaching in medical and nursing schools.

Our National Institute on Alcohol Abuse and Alcoholism (NIAAA) has met with several medical and nursing organizations and will meet with them more formally on the subject in the near future. The goal for these organizations is to take an active advocacy role for increased alcohol training opportunities, including alcohol training during undergraduate and graduate medical education.

Dr. Gordis has met with major health foundations to encourage increased private-sector efforts to improve clinical training on alcohol-related problems for health professionals. I expect this dialogue will continue.

A new "quick response" publication we're getting off the ground should help improve communication between research and clinical practitioners. It's called "Alcohol Alert" -- and its purpose is to get new research findings quickly to clinical providers who can put them into action. This will help primary care physicians and health care workers in a broad array of health care positions, including positions that are not specifically alcohol-related, to increase their understanding of, and to be armed with, the latest technologies in alcoholism diagnosis, intervention, treatment and referral.

Fetal Alcohol Syndrome

Another problem that is or that should be of great concern to the health care professional -- is Fetal Alcohol Syndrome (or FAS).

As a practicing physician I delivered more than 3,000 babies and I have had first hand experience with babies suffering from Fetal Alcohol Syndrome (FAS).

Though not identified as a specific pattern of birth defects until the early 70s, and its incidence and prevalence is not precisely known, there are an estimated one to three cases of Fetal Alcohol Syndrome for every 1,000 live births among the population as a whole. But the prevalence among those who abuse alcohol during pregnancy jumps to an estimated 23 to 29 per 1,000 live births. Fetal Alcohol Syndrome is one of the top three causes of birth defects in the United States today -- and the saddest part is, it's the only one of the top three that is totally preventable.

As a member of the National Commission to Prevent Infant Mortality, I'm especially concerned about the relationship between drinking during pregnancy and infant deaths.

Experts estimate the risk of miscarriages is two times higher than normal for pregnant mothers who drink heavily.

And babies born with FAS are more likely to be of low birth weight -- the major risk factor for infant mortality. FAS babies are 40 times more likely to die in the first month of life. But even if they do survive, such infants often require treatment in neonatal intensive care units and with other expensive technology.

Mental retardation deficiencies associated with FAS are also costly, both in terms of special services needed by those affected and their impaired ability to be productive members of society.

Clearly, both the individual and the society suffer in all of this, but the saddest part is: it's needless suffering. If their mothers had stopped drinking when they became pregnant, the babies could well have been born healthy.

All women need good quality prenatal care, starting early in pregnancy and lasting throughout. They need to know that alcohol consumption during pregnancy is down right dangerous. Screening for alcohol use can be added to the procedures of all providers of prenatal care. If treatment is needed, it can be started immediately and progress monitored throughout the prenatal period.

Most expectant mothers will respond. But it is up to us to give them the opportunity to do so -- by providing them the information they need to ensure a health pregnancy and baby. Some of the materials we've developed to target pregnant women are included in your information packet.

Prevention

Today more attention is being paid than ever before on preventing disease. That includes learning the risk factors involved in the onset of the disease of alcoholism, which can result from the interaction of heredity and environment.

And although there are a growing number of alcohol education programs -- including those based in schools, in communities and those targeting various population groups -- we really need to concentrate our research efforts on determining which programs are effective. In order to encourage the development of new prevention programs, we need to know much more about what really works with different populations.

As a result of my request that the Assistant Secretary for Health study this in greater detail, NIAAA is in the process of developing a new grant announcement which will seek to stimulate relevant alcohol education prevention research.

In addition, I'm pleased to report that our Department's regional health officers, after meeting in February, are examining State and local campaigns to identify and share information on the most successful local efforts.

The National Center for Health Services Research is assisting NIAAA in helping to identify issues surrounding treatment efficacy questions.

Public Education Campaigns

Health messages can play a vital role in increasing public awareness of the linkage between alcohol use and other public health problems such as various cancers, diabetes, hypertension, child abuse, family violence, homicides, and infant mortality. This idea has worked well in our Public Health Service's "Healthy Mothers, Healthy Babies" campaigns.

As you can see from your information packet, we've engaged in an extensive campaign to GET THE WORD OUT. This is the key, whether you're dealing with the Hispanic population, Indians, youngsters, homeless individuals, children of alcoholics, or any other group. We've made many efforts in this regard.

NIAAA is preparing a series of "drop-in" alcoholism and alcohol abuse health messages that will be incorporated into health-related public service announcements, messages and campaigns our Department is sponsoring.

A number of other projects have begun in the area of public education, several involving intergovernmental cooperation or collaboration with the private sector. In your information packet you will find several examples of television, radio and print media projects which have been developed. We've also initiated a series of meetings with other agencies of the Federal Government to see what, if anything, can be done to secure voluntary changes in radio and TV programming to reduce the number of advertisements depicting alcohol use at high risk activities including sports - and to voluntarily change the focus of advertising to adults.

We're also working with private companies in the entertainment and media production industries.

With Hanna-Barbera, the animation company, we're putting together a series of public service announcements on alcoholism and alcohol related problems aimed at children and scheduled for airing on Saturday mornings.

My staff has been meeting with the Advertising Council in New York to explore the feasibility of an innovative new television campaign on alcoholism. We've also agreed to work with the American Broadcasting Company on a major prime-time program devoted to a better understanding of the subjects of alcohol abuse and alcoholism.

The National Broadcasting Company has also met with my staff and is seriously considering making alcoholism and alcohol related problems the centerpiece of their public service campaigns for this January.

Alcohol and Youth

I'd like to turn to the problem of alcohol use among young people. I can't stress enough the importance of getting appropriate messages to our young people. We know that early alcohol use increases the risk for our children's later development of alcohol-related problems.

A statistic I was shocked to learn is that an estimated 4.6 million adolescents in America, or three out of every ten teenagers, experience serious alcohol problems each year. Equally shocking is the fact that nearly nine out of ten teenage auto accidents involve the use alcohol.

And we know that alcohol increases the risk for a range of other problem behaviors -- for example, suicide and homicide -- which affect the very lives of our next generations. One preventable problem that concerns me -- teen pregnancy -- has an alcohol related factor. I've seen studies that indicate that for nearly half of all teen pregnancies one or both of the teenagers involved had been drinking alcohol at the time of conception.

Athletic participation is an important part of growing up for millions of boys and girls in this country. But according to a recent USA TODAY poll of high school coaches, alcohol is interfering like never before in the hopes and dreams of many of these kids. Almost 50% of all coaches surveyed say alcohol use is very serious and getting worse. Reasons most often given for the problem were social acceptance of alcohol, a glut of TV advertising and the ease with which alcohol can be purchased. Drinking is made to seem like a -- quote -- "glamorous and grown-up thing to do," one coach said.

For many years, we have accepted -- or at least tolerated -- alcohol use by older adolescents as an expected "rite of passage" to adulthood. This has got to stop - it is illegal and harmful. In our recent high school senior survey on drug use, we found that almost four in ten seniors reported at least one occasion of heavy drinking in the 2 weeks prior to the survey - heavy drinking defined as a time when they had five or more drinks in a row.

But it's not just high school teenagers who are victims of society's permissiveness and apathy -- younger kids are too.

In one recent survey, an 8th grader was quoted as saying "Beer tastes gross, but I want the effect, so I'll tolerate it." Another said, "Fear of punishment is no good; because we don't believe we'll get caught."

Elementary school kids are tempted to use alcohol too. I was startled by a poll recently that surveyed elementary students' attitudes about wine coolers. Many said they weren't even aware these drinks contained alcohol. That's not really their fault -- and from their perspective, why say no to something that tastes good and you don't view as harmful? For my part I was pretty amazed to find out that there was about the same amount of alcohol in a 12 oz wine cooler as in a shot glass and a half of whiskey.

It has been shown that both alcohol and tobacco are "gateway" drugs -- children and adolescents who use these generally are at greater risk for using other forms of drugs. We know that a youngster who doesn't drink alcohol and doesn't smoke tobacco is much less likely to go on to other drugs.

Many of these problems can be prevented if we teach our children to say no to alcohol and other drugs. If they do - then perhaps they will be able to say no to other problem behavior: and yes to what good things life has to offer them. I'm hopeful that our movement to teach youth to say no to alcohol -- and to give them positive alternatives and positive reinforcement for doing so -- will continue to gain momentum. We need to help them channel their youthful energy and enthusiasm into more worthwhile pursuits, like after-school employment, volunteerism, hobbies, sports, and the arts.

We need this generation more than ever. The American population is aging and our reserve of young talent is shrinking in proportion. In our increasingly competitive world market, we need our youth and we will need them to be bright, alert, flexible, fully educated, and fully ready to meet the enormous challenges of the 21st century.

With responsible peers leading the way, kids can be shown they don't need alcohol to have fun or to make or keep friends.

I'm also hoping that those within our society who interact with youth -- like educators, clergy, coaches, advisors, the medical community and, especially, parents and grandparents, will be persistent in explaining to young people that drinking for them is not only unnecessary, it's both illegal and dangerous.

At HHS, we've made several efforts to combat alcohol use by youth. In addition to the other media campaigns mentioned previously, we're going to continue our "Be Smart! Don't Start!" campaign aimed at 8 to 12 year-olds. And we've just filmed a new television spot aimed at alcohol and other drug use by young people, featuring actor Michael J. Fox, to be aired later this year.

We have initiated education activities targeted to the unique problems faced by children of alcoholics. I believe helping children and families of alcoholics must be part of any realistic answer to alcohol-related problems. That's why I sent Tom Burke to represent me in January at the kickoff of a joint public/private National Elementary School Project we're supporting along with the Metropolitan Life Foundation, geared to the problems of families of alcoholics. Marvel comic books and posters have been sent to all the Nation's elementary schools which will help better educate teachers about how to recognize alcohol problems in families. They'll also alert school children who come from families where alcohol is a problem that there is help available -- for themselves as well as for the family member involved with alcohol.

Our Office for Substance Abuse Prevention (OSAP) is carrying out an aggressive demand-reduction prevention program targeted to America's young people and, in particular, high risk youth and their families. OSAP's programs place a shared emphasis on preventing alcohol use and the use of other drugs by youth.

In the fall of 1987, OSAP awarded 131 demonstration grants to various public and private sector organizations around the country for the development of model, community-based alcohol and other drug prevention programs for high-risk youth. Of these 131 grants, 34 (26 percent) are focusing on an early intervention strategy for those high risk youth who are already using gateway drugs, such as alcohol. In all, about half of the grants have a significant alcohol focus in terms of youthful alcohol use, alcohol use and teenage pregnancy, and children of alcoholics.

Each month, OSAP's National Clearinghouse for Alcohol and Drug Information receives more than 10,000 requests for information from parents, teachers, organizations, businesses, and youths and mails out well over one million pieces of literature a year in response to these requests.

Efforts by the Surgeon General

I've asked the Surgeon General, Dr. C. Everett Koop, to devote some of his talents and energies to my alcohol initiative. He spoke to the National Conference in November and he has some plans of his own in this regard which will begin this summer. Most recently, Dr. Koop held a workshop on health promotion and aging. The first thirty-six recommendations all concerned alcohol issues and the elderly. I am committed to making sure that these recommendations are being carried out.

National Citizens Commission

One of the most successful efforts in the implementation of my initiative is the National Citizens Commission on Alcoholism. I am pleased that Mrs. Betty Ford has agreed to serve with me as honorary co-chairperson. Robert Raclin, our former Deputy Under Secretary is the chair, and Dr. Koop is an honorary trustee.

The goals of the Commission are to focus public awareness on alcohol and alcohol-related problems, stimulate new action in the areas of research, prevention, and teaching, and to advance the National Initiatives.

The Commission membership, I believe is impressive -- and it reflects a wide diversity of citizen leadership from all sections of the country representing the fields of business, health, entertainment, law media, education, and other key groups. A list of the National Citizens Commission trustees is included in your information packet. The inclusion of business, industry and community leaders not previously involved with the field should serve to broaden the base of interest in alcohol problems.

The Future

So where do we go from here?

This fall, we'll be holding our Second National Conference on Alcohol Abuse and Alcoholism with the National Citizens

Commission as our sponsor. Though still in the early planning stages, this gathering will build on the work completed in response to directives received during last November's conference, that we can indeed elevate public understanding about these problems and the initial successes we've had since then.

Our next conference will take a closer look at how alcohol abuse and alcoholism affect other public health problems -- like child abuse, teen pregnancy, homelessness, school dropout rates, physical fitness, and AIDS, as well as such issues as alcohol warning labels and alcohol taxation.

I believe we can elicit change in how people view these problems if we're accurate in our representations and practical in our approach. We need to reach people -- to talk with them in words they can understand and to reach them in familiar settings.

This national agenda is ambitious, but achievable. Achievable only if we work together. The journey ahead is formidable, but attainable. Attainable as we continue our steady course ahead. Let us work together.

Thank you, Mr. Chairman and Members of the Committee.

PREPARED STATEMENT OF THOMAS R. BURKE

Good Morning Chairman Glenn and Members of the Committee,

I am pleased to have this opportunity to discuss with you an area that Dr. Bowen and I, along with others in the Department of Health and Human Services, believe should be a matter of urgent priority to all Americans. That area is alcohol abuse and alcoholism.

Alcohol is the number one drug of abuse in our country. The consequences of alcohol abuse and the disease of alcoholism are among our Nation's most serious health problems. These problems, in fact, exact a far greater impact on our health, social, and economic well-being than the abuse of any other drug.

For too long now we have viewed alcohol abuse and alcoholism, as "shameful" conditions to be hidden and or ignored. A first point in any successful strategy is spreading the awareness that we face major public health problems requiring prevention and treatment.

In addition, alcohol-related problems are devastating to our economy. Costs to the economy were estimated to be $117 billion annually in 1983, the last year that figures are available. These annual costs are expected to grow to $136 billion by 1990 and $150 billion by 1995. By contrast, the estimated costs of illicit drug use and abuse in 1983 was $60 billion.

While a portion of the $117 billion is spent directly by the Government in Medicare, Medicaid and other health and welfare programs, the greatest portion, over $92.8 billion, came from reduced worker productivity and lost employment. This represents products, goods, and services never produced, or delivered. These costs come from reduced productivity, premature loss of life, and accidents (particularly in automobiles), and incarceration of criminals.

The cost of alcohol-related problems also affects our competitiveness in the foreign marketplace. It has been estimated that a significant percentage of the goods produced in the United States are defective and in need of some repair before they even leave the shop. Much of this quality control problem can be traced back to alcohol abuse and alcoholism.

Alcohol-related problems are estimated to be directly responsible for a sizeable portion of the nation's health care costs -- an estimated $14.9 billion annually. Health care costs associated with alcohol abuse and of alcoholism are excessive because alcohol consumption contributes to many very severe medical problems. As a result, alcoholics and heavy drinkers are very high-cost users of health care. Their families are also high-cost health care users. In fact, we know, based on a body of solid research, that the families of alcohol abusers and alcoholics use health care services at twice the rate of those who do not have an alcoholic family member. We also know that treating alcohol abuse and of alcoholism reduces expenditures for overall health care costs not only for alcohol abusers and alcoholics, but for their family members as well.

An area in which we have sent a "mixed signal" is the labeling of alcoholic beverages. We require health warning labels on all sorts of consumer products -- tobacco, certain foods, and other legal drugs -- yet we do not when it comes to products containing the drug alcohol. What does this tell the American public about the potential harmful consequences of alcohol abuse.

As you know, our recent HHS report to Congress concluded that health warning labels do have the potential to affect consumer behavior, particularly in combination with other educational efforts. The Treasury Department -- which has regulatory jurisdiction in this matter -- also has been required by the Senate Appropriations Treasury-Postal Service Subcommittee to undertake a somewhat similar study, which, I believe, is due to be completed at the end of this year. It is our understanding that the Treassury study includes a nationwide public opinion survey on the subject of warning labels.

One heartening development is that Americans appear to have gained a better understanding of the complexity of alcohol-related problems. This can be attributed, in large part, to the many national alcohol organizations that have worked hard for many years to increase awareness of alcohol-related problems as major health problems.

But the fact is, people still do not know all they should. Many do not know how extensive alcohol abuse and alcoholism are and how these problems affect not just individuals, but also families, schools, health care systems, our whole economy and the fabric of our American life. That is why one of the key items in the Secretary's initiative is to mount a concerted educational campaign to heighten awareness.

Private business is concerned as well. They have learned of the economic consequences and are taking action. Approximately 90 percent of the Fortune 500 companies have established employee assistance programs which provide identification and treatment or referral of employees with alcohol-related problems. We in the public sector also have reason for concern. It is estimated that in the Federal workforce alone approximately 3 million federal workers suffer from alcoholism. The benefits of employee assistance programs are substantially fewer sick days and work-related accidents, improved productivity, and lower health care costs.

Small businesses account for approximately 65 percent of the nation's workforce. Clearly, reaching this segment of the population is essential to stem the growing tide of alcohol abuse in America. Therefore, we need to work with small business in a partnership to assist them in developing programs tailored to meet their needs.

Another area targeted in the Secretary's initiative is high risk populations. Specific populations are at high-risk for a variety of alcohol related problems. In 1985, the Department issued its report on the health status of Americans for our four major racial/ethnic minority groups. According to this Report's findings, alcohol-related problems are among the chief causes of illness and death among Black, Hispanic, and Native American populations. A recent study has estimated that as many as one-half of all our homeless in America suffer from alcohol abuse and alcoholism.

Finally, we are looking at ways to fight alcohol abuse among the nation's elderly. Alcohol abuse and alcoholism hit hard this segment of our population -- causing high health care costs paid for by every taxpayer in the United States through Federal health financing programs such as Medicare and Medicaid. Not only are these costs significant now, but, as we are all aware, the elderly population is growing at an enormous pace.

For young, as well as elderly persons on medication, the combination of alcohol and medication may be lethal. By recognizing and treating alcohol abuse and alcoholism, we can cut significantly health care costs. More importantly, we can substantially improve the health and quality of life of Americans.

Early in 1987 recognizing the serious social and economic consequences of alcohol abuse and alcoholism, Secretary Bowen decided to make alcohol abuse and alcoholism priority concerns in HHS for the remainder of his Administration. As a result of recommendation made by the Secretary's panel on alcoholism, he decided on a comprehensive 14-point national initiative.

As Chief of Staff, it is my responsibility to make sure that activities emanating from his announced program are implemented and efficiently managed. HHS is actively pursuing implementation of the initiatives, and I would like to briefly let you know of what has been accomplished to date and what we hope to accomplish over the next several months.

Many ongoing programs in HHS have a natural focus on alcohol abuse and alcoholism. For example, programs which deal with children, youth and families are natural candidates. Health-related research and treatment of such problems as cancer, depression, diabetes, hypertension, child health, and liver disease are other areas where alcohol use and abuse can and should be highlighted. Programs targeted to high risk populations such as adolescents, pregnant women, families without permanent addresses, and the homeless, should address the use -- and abuse -- of alcohol.

We are currently exploring ways in which alcohol abuse and alcoholism can be thoroughly and efficiently integrated into our Department's programs. Secretary Bowen has requested from each Assistant Secretary and Operating Division Head within HHS a summary of ongoing alcohol-related activities for Fiscal Year 1988. The results of our review will be analyzed, and from it we will develop plans to assusre that our existing programs are coordinated to address those areas.

Research is the underpinning of our efforts to combat alcohol abuse and alcoholism. Through research we may be able to develop better procedures for preventing, diagnosing, intervening in, and treating alcohol-related problems. In addition, since we understand that research must be integrated into clinical practice, many of our research and education programs are tailored to meet the needs of the clinical provider. The Public Health Service, acting principally through the Alcohol, Drug Abuse and Mental Health Administration (ADAMHA) and the National Institute on Alcohol Abuse and Alcoholism (NIAAA) serves as the

Federal focal point for alcohol research. They have assumed the major responsibility for developing and implementing a number of the research initiatives.

Dr. Gordis will discuss alcohol research efforts fully. I would like to mention at this time a number of activities in this area which Dr. Bowen did not discuss and that are well underway. These include:

- HHS is cooperating with the American Medical Association to develop a monograph "Alcohol and Drug Abuse: Guide for the Primary Care Physician". Since the primary care provider is on the front line of health care, this information is of critical importance.

- The Indian Health Service (IHS) is conducting a 3-day symposium/training session on alcohol abuse for IHS providers. The IHS will be developing a total of 11 regional resident and treatment clinics for Indian youth with alcohol and other drug abuse problems. The IHS is also is developing an Inservice Continuing Education Program in alcohol and other drug abuse for IHS health care providers.

- A Conference will be held later this year on the topic of alcoholism, - with organizations such as the American Medical Association, the American Association of Medical

Colleges, the American Nurses Association, National League of Nurses and a number of medical and specialty board organizations. The purpose of the Conference is to review the limitations of alcohol content in the curriculum today and the non-emphasis on alcohol-related problems on professional licensure and board exams and, to develop and implement a strategy which will correct these difficulties.

In addition to the activities under way in the Public Health Service, the Office of the Assistant Secretary for Public Affairs has major responsibility for a number of public information/ education activities. Some of the elements of the HHS awareness intitiative include:

- expanding on existing HHS public affairs campaigns and to incorporate alcohol messages in existing campaigns to develop linkages with the private sector;
- embarking on a series of new efforts including proposals to develop high risk group targeted campaigns.

The Department is working closely with the National Association of Broadcasters' - who continue to take a highly visible role in providing alcohol abuse prevention. Among the broadcasters projects are:

- "Project workplace" - a new package of radio and television messages aimed at raising public awareness of health, economic and societal consequences of alcohol and other drug abuse on the job. This should be mailed out to radio and television stations this month.
- Boating safety: new spots are now being developed for summer airing.

Perhaps one of the most exciting results of the Secretary's initiative has been our work with the National Council on Alcoholism to establish a private National Citizens Commission on Alcohol Abuse and Alcoholism. As Dr. Bowen mentioned, invitations to serve on the Commission have been extended to a number of prominent individuals who represent a cross-section of our Nation's public and private citizenry -- representatives from the health field, business and industry leaders, representatives of State and local governments, and the public. A list of Commission Trustees is included in your packet of information. HHS was able to provide funding for initial Commission activities, although private funding for long term operation of the Commission is being sought.

As mentioned earlier, perceptions of these problems is critical. In recognition of this fact, Dr. Bowen has instructed all operating and staff divisions in HHS to discontinue using the term "substance abuse" and to replace it with the term "alcohol and other drugs of abuse". This small but powerful action was unanimously recommended by the planning committee and supported wholeheartedly by the alcohol field.

We do not believe that efforts addressing alcohol issues are or should be the sole province of HHS. Dr. Bowen has therefore sought to bring other Federal agencies and Departments into the process. Examples of cooperative efforts between DHHS and osther Federal agencies include:

- Federal Employee Health Benefits programs now include comprehensive alcoholism treatment benefits due to efforts carried out by the Office of Personnel Management. Dr. Bowen has offered the Department's assistance to OPM in alerting Federal employees to these benefits.

- HHS and the Departments of Labor and Commerce are discussing opportunities for disseminating information on Employee Assistance Programs (EAP) and treatment programs to small businesses and identifying creative ways for small business to join in EAP activities.

- o The Departments of HHS and Education are collaborating in addressing the problem of marketing and promotion of alcoholic beverages on college campuses.

DHHS has initiated dialogues with many Federal, State, local and private agencies to gain their support in promoting non-use of alcoholic beverages for youth and other high-risk persons.

Finally, our second National Conference on Alcohol Abuse and Alcoholism will be held in the Fall of this year. The Conference will be open to the entire alcohol field as well as to interested public participants. There will again be many opportunities for the exchange of information among all elements which comprise the alcohol field -- researchers, clinicians, members of national organizations, and interested citizens. The National Citizens Commission will be a co-sponsor and is planning to hold a "town meeting" when the conference is in session. We welcome the participation of Congress, and hope that you will join with us in bringing to the American people both the knowledge they need to deal with alcohol-related problems and the conviction that together we can make a difference. Let's work together for the betterment of our future, and that of our children. This is a major problem we can beat without massive additional resources if only we provide the national resolve and leadership to do right.

Mr. Chairman, you are to be commended for having the foresight to hold these hearings on the subject that touches the lives of virtually every American. I am encouraged that, working together, great strides can be made to successfully address this national problem.

PREPARED STATEMENT OF ENOCH GORDIS, M.D.

Mr. Chairman and members of the Committee:

Thank you for this opportunity to discuss the alcohol research and other activities we have underway at the National Institute on Alcohol Abuse and Alcoholism. It is a pleasure to be here today.

Demographics

National surveys of drinking practices indicate that approximately one-third of the United States population aged 18 and over are abstainers, one-third are light drinkers, and one-third are moderate to heavy drinkers. Although two-thirds of the adult population drink, actual consumption of alcohol is very unevenly distributed throughout the drinking population. Ten percent of drinkers (6.5 percent of total adult population) who drink the most heavily account for fully half of all alcohol consumed. The other half is consumed by the cumulative 90 percent of the drinking population who are infrequent, light, or moderate drinkers.

According to a 1984 survey, per capita alcohol consumption ranged geographically from a high of 5.34 gallons in the District of Columbia to a low of 1.53 gallons in Utah. Ohio ranked 38th with 2.26 gallons per capita. But let me point out, these figures are approximations because they do not take into account such variables as sales across State lines, home production, illegal production and importation, or the effects of tourism.

How much an individual drinks, how often he or she does it, and what the response to alcohol exposure will be are determined by complex interactions of demographic, social, economic, and biological factors. Age, sex, and ethnic background are significant determinants. Research findings indicate:

- That an estimated 18 million adults 18 years and older in the United States currently experience problems as a result of alcohol use. These may be alcohol dependence symptoms such as binge drinking or loss of control over drinking behavior, or they may be negative personal consequences such as problems with health, work, or personal relationships.

- That people over age 65 consume less alcohol than younger adults, possibly because aging reduces tolerance for alcohol, and they have a lower prevalence of alcohol abuse.

- That women drink significantly less than men and have fewer drinking-related problems, but recently there has been an increase in drinking among women aged 35 to 64 years.

- That Hispanic American men have a higher rate of alcohol use and abuse than the general population. First- generation American-born Hispanics drink more heavily than foreign-born Hispanics. And, nearly half of Hispanic women are abstainers, but less than one-fourth of Hispanic men are. There is a high rate of mortality from cirrhosis among Hispanic American men.

- That American Indians and Alaskan Natives appear to have very high rates of alcohol abuse and alcoholism overall, although many tribes are almost totally abstinent. Liver cirrhosis is the fourth leading cause of death among American Indians.
- That Asian Americans, regardless of national origin, have low levels of alcohol abuse and alcoholism. In fact, abstention is very high among Asian Americans, especially Koreans, Chinese, and women of all Asian groups.
- Abstention from alcohol is more common among blacks than among whites, and black men who drink are less likely than white men to drink heavily. However, black women who drink are more likely than white women to drink heavily. The incidence of alcohol-related medical problems, especially liver cirrhosis and cancer of the esophagus, is very high among blacks. Cirrhosis mortality rates for blacks are twice as high as the

rates for whites.

- That homeless persons in the United States have a high rate of alcohol-related problems.

Scientific research into the courses and consequences of alcoholism and a wide variety of alcohol-related problems has expanded sharply in recent years. In both biomedical and psychosocial areas, alcohol research is now mature and productive. Most significantly, the knowledge being gained in each of these areas increasingly has implications for our understanding -- and deeper exploration -- of the other.

Genetics

Evidence for genetic predisposition to alcoholism continues to grow. It is now widely accepted by researchers in the field that alcoholism can result from the interaction of heredity and environment.

Studies have been conducted of individuals who had a biological parent with alcoholism but were removed from the alcoholic environment through adoption at an early age. The studies allowed assessment of the relative contribution of genetic and environment factors in the genesis of alcoholism. Through these studies, two types of genetic predisposition to alcoholism have been identified, male-limited and milieu-limited.

- Male-limited susceptibility occurs only in males, is highly heritable, gives rise to severe early-onset alcoholism often requiring extensive treatment, and is associated with serious lawbreaking.

- Milieu-limited susceptibility, which was found to occur in both sexes, is perhaps involved in most cases of alcoholism. This type of hereditary alcoholism has a late onset, is usually not as severe as male-limited alcoholism, and is not associated with the legal system. Milieu-limited susceptibility requires environmental provocation to become expressed as alcoholism, but environmental provocation does not include alcoholism in the adoptive parents. The only significant parental factor found associated with this type of alcoholism in the adoptees was low socioeconomic status of the adoptive father.

Further evidence for the operation of genetic factors in alcoholism has come from selective breeding experiments. Animal lines have been produced that differ in susceptibility to various effects of alcohol on the central nervous system, including different susceptibility to alcohol tolerance and severity of withdrawal symptoms. Lines have also been bred that differ in their rates of alcohol metabolism. Finally, selective breeding has yielded alcohol-preferring and alcohol-nonpreferring lines.

The ability to produce such animal lines is excellent evidence that susceptibility to important effects of alcohol can indeed be inherited.

Genetic molecular variations in alcohol-metabolizing enzymes are a major area of research on the heredity of alcoholism because a mutation that produces a slight alteration in the molecular structure of these enzymes could be expected to have a pronounced effect on their ability to remove alcohol from the body. Many investigators believe that such studies have the potential of explaining fundamental mechanisms of alcoholism and of identifying genetic markers of susceptibility.

There is considerable evidence that Oriental populations have a very high prevalence of a form of aldehyde dehydrogenase that has a low efficiency in removing acetaldehyde, a toxic compound that is the first product of alcohol metabolism. Rapid accumulation of acetaldehyde, after only a few drinks, produces skin flushing, a rapid pulse, and other uncomfortable symptoms, and this discomfort may deter further drinking. The high prevalence of the gene for this form of the enzyme in Oriental populations may help explain their low rates of alcoholism.

The Institute supports fundamental studies in genetics and molecular biology to understand heritable biochemical and physiological factors that can affect susceptibility to alcoholism and other consequences of alcohol use. Such studies include research on the molecular genetics of alcohol metabolizing enzymes in both animals and humans, investigations of hereditary variations in the molecular properties of nerve cell membranes and how these variations affect responses to alcohol, genetic factors that modify alcohol's effects on neurotransmission, and gene-environment interactions in the development of alcoholism.

In 1987, the Institute began a major initiative to promote research on the genetics of alcoholism by inviting applications to establish a new National Alcohol Research Center devoted to studies in this area. As a result, a grant to establish the Center was awarded to Indiana University School of Medicine.

Neurosciences

The brain is the site of many of the most immediate and obvious effects of alcohol consumption, as well as the site of many of the serious effects of chronic alcohol abuse. The signs of acute intoxication, tolerance, physical dependence, and withdrawal all involve interactions of alcohol with neurons of the brain, but the molecular mechanisms of these effects are only beginning to be understood.

One of the difficulties of studying alcohol-brain interactions is that there appear to be no specific receptors for alcohol on cell membranes of the central nervous system. This makes the study of these interactions much more difficult and complex for alcohol than for other psychoactive substances, such as opiate drugs, for which specific receptors have been identified. Instead of binding to a specific receptor site, alcohol diffuses into the lipids of the nerve cell membrane in a manner that is not highly site-specific. As a result of this generalized penetration, the lipid molecules lose some of their organization and the membrane loses some of its rigidity. This disordering, or "fluidizing", effect modifies the function of certain proteins embedded in the membrane that are involved in critical nerve cell functions, including the transmission of nerve impulses. The membrane disordering effect of alcohol in nerve cells is currently the most generally accepted hypothesis regarding alcohol's effects on the brain.

The potential benefits of understanding such fundamental mechanisms are great. Among the possible benefits are the identification of biochemical markers indicating unusual risk of alcoholism, as well as better methods of treating established alcoholism. Such research might someday lead to the design of specific pharmacological interventions that could reverse the molecular alterations produced by alcohol in nerve cells.

Medical Toxicology

It has long been believed that practically all alcohol is absorbed from the gastrointestinal system and is metabolized mostly in the liver. However, new evidence indicates that alcohol metabolism also occurs in the lining of the stomach. Gastric alcohol metabolism may provide protection against toxicity when moderate amounts of alcohol are consumed. Nevertheless, alcohol consumption can cause gastric lesions, especially when taken with other substances that irritate the stomach, such as aspirin. Alcohol can also affect the small intestine by altering intestinal motility, metabolism, blood circulation, and cellular structure, and it may also cause malabsorption of nutrients.

Most evidence indicates that development of liver cirrhosis requires the chronic consumption of large amounts of alcohol. However, newer studies suggest an increased risk in men who drink as little as three drinks a day and in women who drink as little as one and one-half drinks a day. Recent studies also indicate that alcohol has an adverse effect on heart tissue even in young adults. And, several studies indicate that alcohol consumption is associated with hypertension and blood coagulation disorders.

Alcohol has major effects on the endocrine and reproductive systems in both men and women. Chronic heavy alcohol consumption in men reduces the levels of testosterone and some alcoholic women develop menstrual disturbances, infertility, and hormonal imbalances. Alcohol has also been shown to have major effects on the levels of thyroid hormones, and there is some evidence that such alterations may be involved in the development of a type of liver necrosis in chronic alcohol abusers.

Further, chronic alcohol abuse is associated with increased susceptibility to infectious diseases and several kinds of cancer.

Although our understanding of the medical consequences of alcohol continues to grow, there are many unanswered questions. A major need is for study of the fundamental cellular mechanisms that can explain the enormous range of alcohol's effects throughout the body.

Fetal Alcohol Syndrome

We have come a long way in our understanding of the condition known as Fetal Alcohol Syndrome (FAS). Wide recognition of the FAS came in the late 1960s and early 1970s with the publication of case reports from France and the United States.
A decade-long research effort investigating the effects of alcohol on pregnancy has conclusively established that alcohol use during pregnancy poses a threat to the health of the child. FAS is a serious combination of birth defects involving both physical and mental impairments. It is estimated that today between one and three babies in every 1,000 live births in American families are seriously affected, and between three and five children per 1,000 are partially affected by FAS. The Public Health Service is committed to reducing the incidence of

FAS by 25 percent by 1990.

In the meantime, substantial progress is being made in understanding the effects of prenatal alcohol exposure on the developing organism. Specific brain structures are being identified that are especially sensitive to alcohol and are involved in functions known to be impaired in the fetal alcohol syndrome. Knowledge of the subtle effects of maternal social drinking on the developing infant is growing. Factors that modify the risk of an alcoholic woman giving birth to a baby with the fetal alcohol syndrome are being discovered. Stages of pregnancy at which the embryo or fetus is especially vulnerable to alcohol are being identified. Scientific attention is being given to subpopulations that appear to be especially vulnerable to alcohol-related birth defects, an area of inquiry that can lead to more effective prevention efforts. And increasing new leads are being explored to understand underlying mechanisms.

Treatment Research

In recent years, the maturing field of alcoholism treatment research has focused increasingly on three major domains: (1) defining the types of alcoholic patients; (2) defining appropriate treatments for specific kinds of patients; and (3) improving the technology of treatment for all patients.

Research on defining the types of alcoholic patients is proceeding in two areas: (1) genetic analysis, and (2) clinical observations, which classify alcoholics on the basis of such factors as severity and type of clinical complications, quality and quantity of the patient's economic and social supports, and presence or absence of psychiatric problems separate from the drinking problem itself. A major need is to synthesize the genetic analytical and the clinical observational approaches, since clinical indicators of alcoholism severity may also have a genetic basis.

Treatment approaches for alcoholism are of two general kinds: verbal and pharmacological. Verbal treatments make extensive use of various forms of individual counseling and group therapy. They can range from minimal interventions, such as a physician's warning to a patient who is drinking too much but has not yet progressed to physical dependence, to more formal and intensive counseling involving training in life skills, relapse prevention, stress reduction, and involvement of support groups such as Alcoholics Anonymous. In contrast to classical insight-oriented psychotherapy, alcoholism counseling is directive, supportive, reality-centered, focused on the present, and oriented toward changing behavior. Counselors and therapists of various therapeutic perspectives play a crucial role in the treatment of alcoholism.

Pharmacological therapy for alcoholism has been available for some time but has yet to achieve its full potential. The traditional pharmacological treatment for alcoholics has been disulfiram (Antabuse), a drug that provides deterrence to relapse by interacting with alcohol metabolism to cause a very unpleasant reaction. The drug, taken voluntarily or in some cases under judicial compulsion, has long been used as an adjunct to more comprehensive alcoholism treatment programs, but its effectiveness in promoting abstinence has been questioned recently. One group of investigators has reported that disulfiram made no contributions to sustained abstinence, delay of relapse, employment, and social stability beyond those provided by counseling alone, although the total number of abstinent days in the treatment group was higher. Some alcoholism treatment experts suspect that the willingness to take disulfiram daily may only reflect a strong motivation to stop drinking, and that those who are willing to take the drug would probably succeed in quitting without it.

Increasing knowledge of the neurochemistry of alcoholism has opened the possibility of developing drugs that work directly to counteract neurochemical mechanisms involved in the alcoholic's craving for alcohol. This is a very promising area of research in alcoholism treatment because it attacks the problem at its neurophysiological core. Among potentially useful drugs being explored in this category are those that inhibit serotonin uptake by brain nerve cells having receptors for this neurotransmitter, which is directly involved in the alcohol craving phenomenon.

Investigators are testing various alcohol treatment methods for effectiveness and reliability to devise methods to match persons seeking help for alcohol-related problems with the optimal treatment approach and setting.

For example, scientists at the National Alcohol Research Center at the University of Connecticut are developing a multi-dimensional classification system of persons suffering from alcoholism that can be used to help predict treatment outcome for inpatient treatment services. To do this, investigators are gathering and analyzing information on family histories, drinking patterns, and social adjustment, among other factors, and comparing the data with treatment outcome. This typology strategy helps assess types of patients who can best benefit from a specific treatment. Research of this nature has the potential to improve treatment outcome, cut medical costs, and provide guidelines for public policy decisions regarding third-party reimbursement.

Excellent progress is now being made in research laboratories as a result of an NIAAA initiative to develop an objective chemical marker of chronic drinking which would be informative even if on the day of sampling the patients were abstinent. This technology will have wide impact on evaluation of treatment, epidemiology, and clinical diagnosis.

I see treatment research as such an important area that in a recent reorganization at the Institute, I created a Division of Clinical and Prevention Research which includes a Treatment Research Branch.

Further, as mandated by the Congress, the NIAAA has commissioned a study of alcoholism treatment by the National Academy of Sciences. The NAS will (1) review available knowledge and experience regarding the effectiveness, availability, costs, quality and appropriateness of treatment for alcoholism and alcohol abuse, and (2) provide recommendations on policies and programs of research, planning, administration, and reimbursement for treatment and rehabilitation of persons suffering from alcoholism and alcohol abuse. The final report of the study will be submitted by NAS to the Secretary and transmitted by the Secretary to Congress in mid-1989.

Prevention Research

The goal of the Institute's prevention research program is to increase knowledge about the effectiveness of alternative measures to reduce the incidence and prevalence of alcohol-related problems. Prevention strategies can be considered in two general categories -- (1) education and communication and (2) laws and regulations. Research indicates that the complexity of factors influencing drinking behavior, as well as the wide range of alcohol-related problems, requires combining various prevention strategies to address both individuals and their environments.

Recent studies indicate that mass media campaigns to educate the public about the risks of alcohol abuse and alcoholism are not sufficient by themselves to alter health behavior. Instead, their greatest utility appears to be in creating a context that allows other, more targeted strategies to succeed. For example, there is evidence that mass media public information campaigns increase the effectiveness of efforts such as stricter enforcement of drinking-and-driving laws, community-based programs, and focused health promotion campaigns.

So far, scientific evidence of effectiveness in preventing alcohol-related problems is strongest for such measures as increasing the price of alcoholic beverages, raising the minimum drinking age, and increasing the probability of detection and punishment for drinking and driving. A number of studies have found that the relative price of alcohol influences both the alcohol consumption level and the incidence of certain alcohol-related problems. In several States, a pronounced decline in the number of automobile accidents and fatalities involving young people has been attributed to legislation raising the minimum drinking age.

Further growth in our understanding of alcohol abuse and alcoholism will require the study of genetic factors, neural organization, emotion, social and cultural influences -- and the interaction of all these elements with one another. The questions are complex and elusive and certainly among the most difficult in science. We have outlined for you today the tremendous progress made in this field over a relatively short period of time and look forward to even greater successes in the future.

That concludes my testimony. Thank you for inviting me. I will be glad to answer any questions you may have concerning this subject.

NASADAD

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TESTIMONY OF

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DIVISION OF ALCOHOL AND DRUG ABUSE
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and

IMMEDIATE PAST PRESIDENT

NATIONAL ASSOCIATION OF STATE ALCOHOL AND
DRUG ABUSE DIRECTORS

before the

COMMITTEE ON GOVERNMENTAL AFFAIRS
UNITED STATES SENATE

on

FEDERAL AND STATE SUPPORT FOR ALCOHOL ABUSE SERVICES

MAY 25, 1988

INTRODUCTION

Good morning Mr. Chairman and Members of the Committee. Thank you for the invitation to appear before you today to address the role of the States in providing alcohol prevention and treatment services.

My name is Anne D. Robertson. I serve as Director of the Mississippi State Division of Alcohol and Drug Abuse within the Department of Mental Health and am appearing before you today as the Immediate Past President of the National Association of State Alcohol and Drug Abuse Directors (NASADAD). NASADAD is a not-for-profit organization whose membership is exclusively comprised of the State officials designated by the Governors to administer the publicly-funded alcohol and drug treatment and prevention system.

Before I begin our statement, I wish to express our sincere appreciation and recognition of your interest and leadership in holding this series of hearings to address alcohol abuse and alcoholism prevention, treatment, research, and policy issues.

Mr. Chairman, you are already familiar with the range complexity and seriousness of the problems that result from alcohol abuse and alcoholism. These problems impact on every sector of our society transcending socio-economic levels, race, age and sex. Nearly 96 million men and women - more than two-thirds of the nations's adult population - use alcoholic beverages. Most have learned to use these beverages without ill effects. However, about 10 million people have become problem drinkers or alcoholics. Alcoholism affects more than one-third of our country's families; children raised by alcoholic parents are four times more likely to become alcoholics than children from non-alcoholic homes; adult children of alcoholics are twice as likely to experience depression or other mental health problems. While the impact on individuals is often tragic, the costs to their unborn children, their families, and to society as a whole are even more staggering.

What makes the alcohol problem even more alarming is its impact on young people. Drunken driving is the leading cause of death among those fifteen to twenty-five years of age. In many schools, alcohol & drug use has reached epidemic proportions. A children's magazine found that one-third of the fourth graders it surveyed reported "some" to "a lot" of pressure to drink alcoholic beverages. And student leaders report that teenage alcohol use is the single biggest problem facing schools.

HISTORICAL PERSPECTIVE

The publicly-funded alcohol prevention and treatment system relies on financial support from all levels of government - Federal, State and local. This partnership effort has evolved over the past two decades with the roles and responsibilities of the various government entities changing over the years. The

Federal government's initial efforts in the alcohol abuse field were to provide national leadership and to stimulate State and local government participation in the development of a well-coordinated and comprehensive alcohol service delivery system.

The State Alcohol Agencies and the Single State Agencies for Drug Abuse Prevention were initially created by the States in response to Congressional Action on the Comprehensive Alcohol and Alcoholism Prevention, Treatment and Rehabilitation Act of 1970 and the Drug Abuse Office and Treatment Act of 1972, respectively, to have sole responsibility in the State to plan and administer a Statewide alcoholism and/or drug abuse prevention and treatment network.

At that time, State Alcoholism Agencies and State Drug Abuse Agencies were generally small, had limited authority and low visibility within the State governments and State and local financial support of community-based service programs was very limited with the notable exception of a few States. However, with encouragement from the Federal government, the States soon assumed a significant portion of the responsibility for allocating and monitoring not only their own State revenues for alcohol abuse services, but also for awarding and monitoring Federal dollars to entities within the States for alcohol-related services and programs, thereby permitting the States to allocate the dollars where they were most needed.

Under the Omnibus Reconciliation Act of 1981, which created the Alcohol, Drug Abuse and Mental Health (ADMS) Block Grant and significantly revised the role of the National Institute on Alcohol Abuse and Alcoholism (NIAAA) and the National Institute on Drug Abuse (NIDA), the mandate for these State structures was repealed. However, I am pleased to report to you that each State and U.S. Territory has retained the single State agency governmental structure to effectively coordinate State alcohol and drug abuse treatment and services, including administration of the relevant portions of the ADMS Block Grant.

When the ADMS Block Grant was authorized in 1981, the responsibilities of the Federal government for the administration of the Federal alcohol and drug abuse services programs were eased significantly. The ADMS Block Grant, authorized by P.L. 97-35, turned complete administrative responsibility for the Federal alcohol and drug abuse service dollars to the States. Already responsible for administering three-fourths of the Federal alcohol and drug abuse programs through a mechanism similar to the ADMS Block Grant, the States were well prepared and willing to accept responsibility for awarding and monitoring the service dollars included in the ADMS Block Grant program.

The transition from categorical to block grants for the State alcohol and drug abuse agencies was smooth and did not cause major disruption in services due in part to a temporary overlap of Federal funding. All of the 50 States and the U.S.

Territories participated in the ADMS Block Grant program during its first year - Fiscal year (FY) 1982 - and have continued in the program ever since with no major problems arising which relate to the ADMS Block Grant as a funding mechanism.

Since the State alcoholism and drug abuse agencies were already administering three-fourths of the programs eventually folded into the alcohol and drug portion of the ADMS Block Grant Program, the basis for the supposed administrative cost savings associated with the ADMS Block Grant - streamlined and efficient management - was not applicable. The funding reductions accompanying the Block Grant were supposed to be balanced by the increased costs savings realized from the new funding and administrative mechanisms. Unfortunately, for the alcohol and drug services programs the supposition was not relevant. Rather they were penalized for having been one step ahead.

ALCOHOL SERVICES UNDER THE BLOCK GRANT

A major continuing cause for concern and one which has hampered many States' abilities to provide alcohol services to those in need has been the drastic reduction in Federal financial support which accompanied the ADMS Block Grant program. In FY 1980 (the base year for the alcohol and drug portion of the ADMS Block Grant) Federal appropriations for the alcohol and drug abuse project and formula grant programs totaled $332 million. In FY 1988 the alcohol and drug portion of the Block Grant equals only $243 million - a substantial reduction from FY 1980 levels without adjusting for inflation. If inflation rates from FY 1980 through FY 1988 are taken into account, current Federal funding levels for alcohol and drug treatment and prevention services represent an even greater decrease.

Exhibit A included with my written statement provides a comparison of Federal appropriations for alcohol and drug treatment and prevention services from FY 1980 to FY 1988. This comparison demonstrates significant reductions in Federal support. Only in FY 1987 and FY 1988 are increases in support documented. This is due to enactment of the Anti-Drug Abuse Act of 1986 (P.L.99-570) which authorized a supplemental, emergency grant program for treatment services with the majority of these new funds supporting drug abuse treatment services.

When the level of Federal support for alcohol and drug treatment and prevention services is adjusted for inflation, as displayed in Exhibit B, it is discouraging to note that even the FY 1987 and 1988 levels of support are clearly below the level of Federal monies needed to maintain the level of services supported by the Federal government eight years ago.

Accompanying this lack of Federal financial resources for alcohol prevention and treatment programs is the inability of each of the States and U.S. Territories to meet the demand for services. In an annual survey conducted by NASADAD, each State agency director is asked to provide a narrative description of major needs for

Comparison of Federal Appropriations for Alcohol and Drug Treatment Services From FY 1980 to FY 1988

($ in 100,000 and not adjusted for inflation)

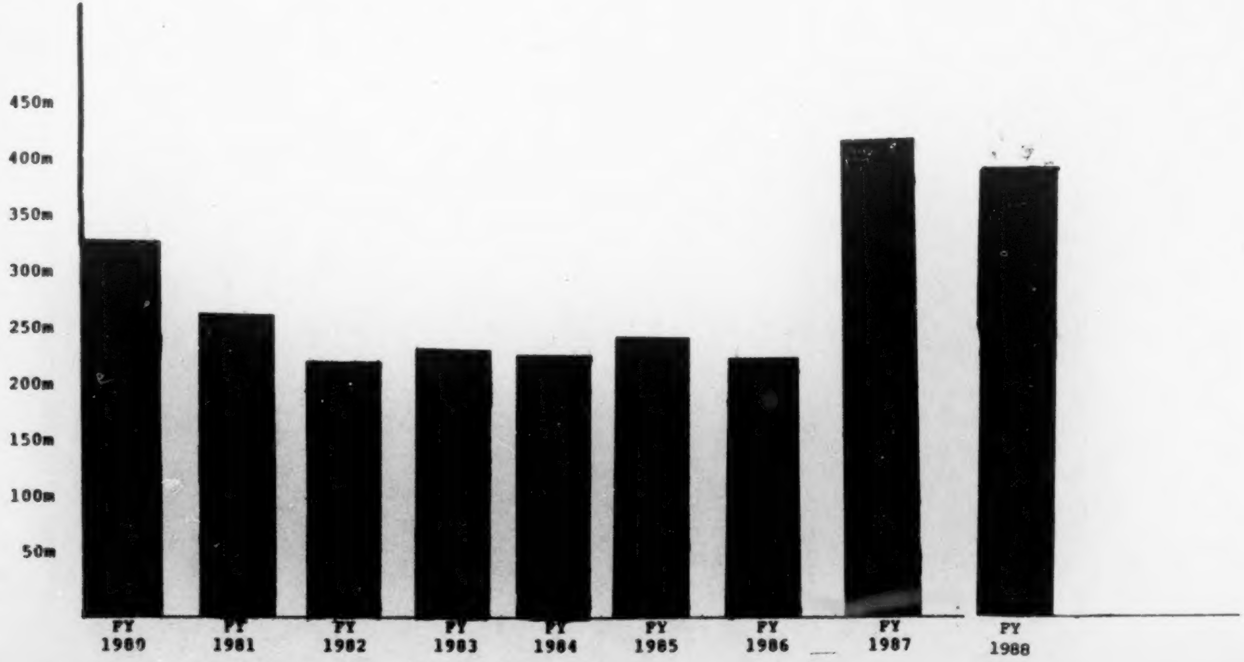
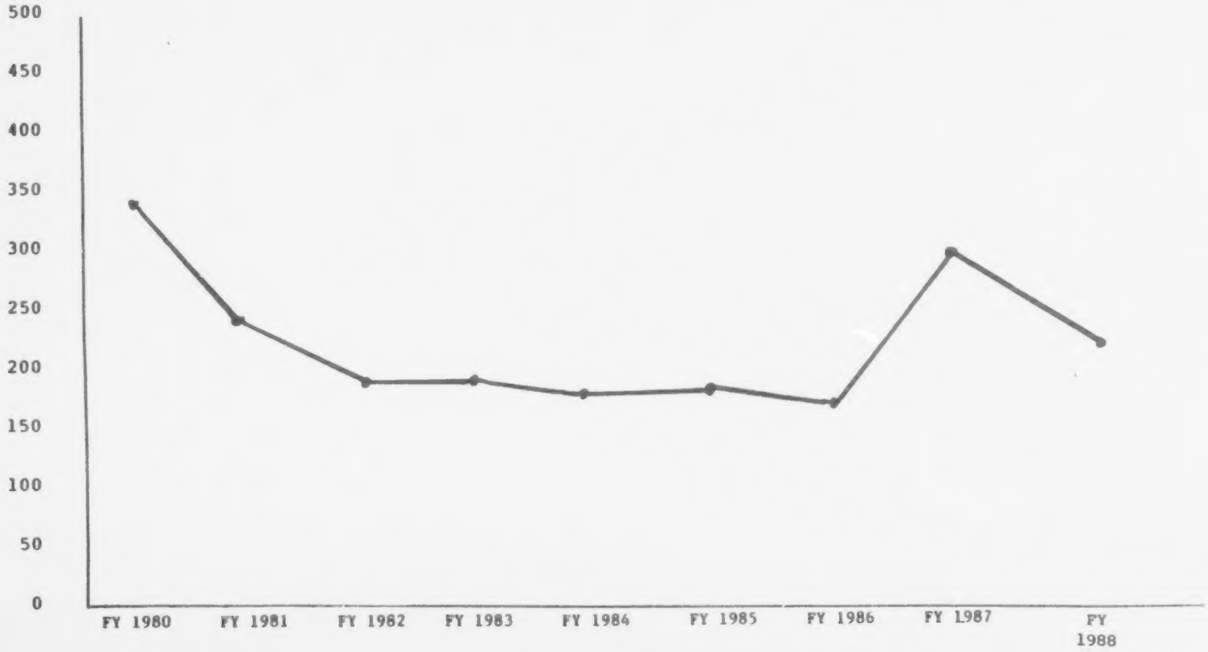


EXHIBIT B

Comparison of Federal Support
for Drug and Alcohol Treatment
Services in Selected Years
and Adjusted for Inflation*



* Based on official CPI estimate from Department of Labor

which resources are not adequate. In the most recent survey on Fiscal Year 1986 resources, the State responded that major needs regarding alcohol services include the inability to meet the requirements of special populations, including adolescents, women, minorities and the elderly; the lack of adequate detoxification services; insufficient outpatient programs; and the need to increase staff positions and salaries. Some States also indicated other needs including long-term shelters for homeless alcoholics; lack of outreach services in rural towns and communities; and the need for additional facilities and staff to service the backlog of clients awaiting treatment.

As mentioned previously, the Anti-Drug Abuse Act of 1986 authorized a new block grant for alcohol and drug abuse treatment and rehabilitation (ADTR) services. This ADTR Block Grant provided $163 million to the States in FY 1987 and $156 million in FY 1988. Although the legislation permits the use of these funds for alcohol-related services and many States have chosen to expend some of these funds for alcohol abuse treatment and rehabilitation, the States have come under criticism for doing so. Since the focus of both the Administration and Congress is on the abuse of drugs other than alcohol, they have encouraged the States to use the majority of the ADTR monies for anti-drug abuse activities. Thus, in spite of the additional of new Federal funds, the Federal government has not encouraged expansion of services to meet the needs of those suffering from alcohol abuse and alcoholism.

The ADMS Block Grant permits each of the States to assess individual State needs and to use Federal funds to meet those identified needs. States are required to submit plans detailing the proposed uses of the Federal block grant funds and to comply with the mandates of the Block Grant, i.e., using 35 percent of the funds for alcohol services; 35 percent for drug services; 20 percent for prevention; 5 percent for services for women; and a 2 percent limit on the use of block grant funds for administrative expense. Additionally, none of the Federal funds may be used for capital construction or renovation of facilities.

While the States were previously required to report data to the Federal government as a condition of receipt of Federal funds, these requirements were eliminated with the authorization of the ADMS Block Grant. The States do not wish to return to the days of mandated reporting requirements which collected data, the value and usefulness of which was at times questionable. However, the States and Institutes currently need support for the development of a mechanism and process by which significant data can compared with other States across the nation or even within their own regions.

States have become increasingly concerned over the lack of data available at the national level which comprehensively and accurately describe the treatment and prevention network and its clients. While a considerable amount of data was collected by the National Institute on Alcohol Abuse and Alcoholism prior to the beginning of Fiscal Year 1982, only limited data has been collected since that time period. In the meantime, States have sought to revise their data collection systems to make them more responsive to their individual needs. As a result, the ability of States to compare information based on the same data elements and definitions is being threatened. As responsible State officials, we believe that a uniform data collection tool and process should be jointly developed and implemented by State and NIAAA representatives with Federal financial support. NASADAD is willing to work with the Congress and the Secretary in whatever way we can to assist in enhancing national data collection efforts which will help the alcohol field to better assess the service delivery system and its clients.

In closing, we encourage the Committee to recognize that the publicity-funded alcohol and drug abuse services programs which rely on Federal, State and local government funds remain effective providers of low-cost quality services. The State Alcohol and Drug Abuse Agencies are pleased with the ADMS Block Grant program and its accompanying philosophy that the States are able to identify the needs of their citizens and to determine where Federal support will be of most value. However, without an increase in Federal financial support and leadership, we expect to see a continuing deterioration of the alcohol abuse services system. We would like to once again express our appreciation for your active interest in the alcohol abuse field and encourage the support of your committee in seeking ways to build upon the Federal-State partnership.

Thank you.

5

An Overview of Federal Alcohol Policy

Statement of

Christine Lubinski

on behalf of

National Council on Alcoholism, Inc.

before the

Committee on Governmental Affairs

United States Senate

May 25, 1988

NCA is a private nonprofit health organization

My name is Christine Lubinski and I am the Washington Representative for the National Council on Alcoholism. The National Council on Alcoholism is the leading voluntary health organization working nationwide to prevent and reduce alcoholism, drug dependency and other alcohol and drug-related problems. NCA's two leading missions are public education and public advocacy. We have 190 state and local affiliate councils which provide information and referral services to families and individuals with alcohol and other drug problems. Many of these local councils conduct prevention and education programs and involve citizens in grassroots advocacy efforts.

NCA is delighted to see the interest of this Committee and its Chairman in federal alcohol policy. NCA was founded in 1944 to educate the public about alcoholism as a treatable disease and to advocate for services and public policy in the best interests of alcoholics and their family members. NCA has long been on record in support of strong federal leadership to combat alcoholism and alcohol-related problems. Alcoholism and related problems constitute a major public health threat to the nation.

The history of the federal alcoholism effort begins in earnest with the enactment of the Hughes Act in 1970, a federal law initiated in the United States Senate which resulted in the creation of a network of public treatment services and which brought recovery from alcoholism within the reach of thousands of Americans. Without the national leadership framed in the Hughes Act, a characterization of alcoholism as a moral weakness might still dominate the public mind. We remain forever grateful to Senator Hughes and his work in creating the National Institute on Alcohol Abuse and Alcoholism.

From the start, NIAAA had a stepchild status, underscored by its first bureaucratic placement in NIMH. Throughout its history it has always been low institute on the funding totem pole following mental health and drug abuse. NIAAA managed to get out from under the wings of NIMH in 1973 and become a co-equal institute with NIMH and the newly created National Institute on Drug Abuse under the administrative superstructure of the Alcohol, Drug Abuse and Mental Health Administration (ADAMHA).

In those early days, NIAAA funded treatment services through direct grants to community organizations, the project grant program, and to states through the formula grant program. The constituency supporting NIAAA became stronger with the growth in funding from $14 million dollars in 1971 to nearly $200 million in 1974 with the release of impounded monies.

Because of a strong constituency and advocates on Capitol Hill, NIAAA was able to hold its own in the face of constant efforts within HEW to erode its status as an independent Institute, to move it to NIH, to fuse it with NIDA and to cut its staff and drastically reduce its funding.

The first real blow to NIAAA as the focal point of the federal alcoholism effort came with the Omnibus Budget Reconciliation Act of 1981. Treatment services were stripped from the Institute and moved into the alcohol, drug abuse and mental health services block grant administered by ADAMHA. Treatment funding was reduced by 40% Many public treatment providers argued that the demise of direct federal support for services came five years too early to ensure stability of the system. We lost valuable information about treatment as federal data collection efforts came to an abrupt halt. During the same period, NIAAA lost its clinical training program, which was used primarily for the Career Teachers Program -- designed to inculcate alcoholism as an integral part of the curricula in medical schools. NIAAA was relegated to a small research institute with some adjunct prevention and research training activities and a funding level of $20 million.

Despite this dramatic and abrupt reduction in federal oversight and support, there is a general consensus in the field that the public treatment system has survived, if not thrived, in this interim period. Local advocacy in many states generated increased state financing for services. But what of the losses? They are immeasurable and include the loss of national knowledge about the numbers of individuals seeking public treatment services, their demographic characteristics and the outcome of their treatment. New Federalism hit NIAAA just as federal treatment standards were being developed. None now exist. And what of underserved and unserved populations? NASADAD's annual survey of state alcohol and drug agencies suggest significant gaps in public treatment services. Appropriate treatment for women and their children, adolescents, and the long term care needs of homeless alcoholics are frequently cited by states as priority populations if new funding becomes available.

Gaps in information about the public treatment system, partially financed by federal dollars, has had its political as well as clinical and practical ramifications. There is no federal ownership for a block grant that many see as little more than a revenue sharing program This lukewarm federal commitment was evidenced by base level appropriations year after year until the

Anti-Drug Abuse Act of 1986 breathed some new life into the block grant program.

In 1984, NCA successfully advocated for federal leadership to generate new and expanded services for women with alcohol and other drug problems from block grant dollars. It is no secret that reductions in funding for health and social service programs tend to disproportionately affect women and other underserved populations -- a last funded, first eliminated phenomenon. Initiatives began by NIAAA in the seventies to educate the treatment constituency about the special outreach and treatment needs of women through technical assistance and financing of model programs to provide services to women had all but disappeared under the block grant. States were resistant to any attempt to direct the expenditure of block grant funds and many members of our own alcoholism constituency viewed initiatives for special populations, especially women, as an outmoded idea of the seventies, no longer applicable in the current climate.

NCA's view then as well as now is that the Federal government has a responsibility to assess the national scene, identify unmet needs, and encourage or mandate the expenditure of federal dollars to meet the needs of unserved or underserved populations. Now that federal block grant dollars represent only twenty percent of all public money spent on treatment, national interests can best be served by directing those federal dollars to provide services for the underinsured and the uninsured as well as those with particular cultural, ethnic or gender needs. This federal direction is critical if the treatment needs of all populations -- women, ethnic and racial minorities, adolescents, the homeless and the indigent -- are to be met. It is also a viable political strategy to humanize the block grant by recognizing special populations and by doing so, to maintain a still needed federal commitment to the provision of treatment.

In the meantime, while constituency efforts continued to support a federal role in the provision of treatment through the block grant, NIAAA slowly increased its research budget as it simultaneously narrowed its focus. The Institute which once had garnered broad based support from an alcoholism constituency comprised of volunteer activists, counselors, treatment providers and prevention advocates became a primary interest only of the research community itself. Although NCA continues to advocate for increased funding and a broadened focus for the Institute, neither constituency groups nor policymakers view NIAAA as a force in alcohol policy or the daily work of prevention and treatment professionals in communities across the nation. In 1981, when the Office of Personnel Management facilitated the withdrawal of the alcoholism treatment benefit, the Institute was not asked nor did it choose to comment. When Congress deliberated the "21" drinking age legislation as an initiative to reduce alcohol-related fatalities and other alcohol problems among youth, NIAAA played no role. When alcohol advertising, health warning labels on alcoholic beverages and increased taxes on alcohol entered the national debate as public health strategies to reduce alcohol problems, NIAAA was silent and only infrequently viewed as a resource for policymakers or advocates on these issues.

In fact, Congress moved to further reduce NIAAA's role in 1986 when it removed virtually all prevention activities from the Institute and placed them in the newly created Office for Substance Abuse Prevention (OSAP). Congress further limited alcohol prevention by omitting the word alcohol from the new federal haven for prevention and then virtually restricting OSAP activities to so called "high-risk youth." We live in a culture in which the pervasive availability and marketing of alcoholic beverages creates a significant level of risk for anyone and everyone who chooses to drink. However, concerns and questions raised by NCA and others about the mandate of OSAP as well as the overall short shrifting of alcohol in the debate and content of the omnibus drug initiative were overshadowed by the national attention to the war on drugs and the biggest funding boom to the cause since the beginning of the Reagan era.

After all, it was difficult to complain. Alcohol and other drug research and treatment efforts continued to experience healthy increases in funding, particularly relative to other health and social service programming. It was equally difficult to publicly criticize the narrowing biomedical focus of NIAAA when it was clear that such a focus was a critical key to survival and growth for the Institute. Psychosocial research was in general disrepute, data collection viewed as undue interference in local and state activities, and prevention research -- which might offer new challenges to the powerful alcohol beverage industry and dramatic new gains in public health efforts to reduce alcohol problems -- were simply not on this Administration's agenda.

The notable absence of attention to alcohol, the nation's most popular and costly drug, in the Anti-Drug Abuse Act of 1986 was also difficult to counter. Alcohol was included in most of the prevention and treatment components of the bill, if only mentioned obliquely in the debate and the statutory language. Furthermpre, the debate so frequently teetered on the brink of a war on drug users instead of a war on drugs that advocates were hard pressed to offer alcoholics up as additional victims in the fray. And almost ignored has been the fact that now in all 50 states, alcohol is an illegal drug for persons under 21. Finally, the roughly $600 million in prevention, education and treatment funding was a sufficient sweetener to the $1 billion in military , law enforcement and other drug interdiction efforts to please a constituency desperately in need of those dollars while concerned about the vitriolic and hype of the overall effort.

The need for a comprehensive strategy to reduce both the supply of and the demand for illegal drugs has been strongly resisted in policy discussions about the prevention of alcoholism and other alcohol-related problems. It is striking that recommendations calling for invasions of third world countries and increased deployment of the military to control the supply of illegal drugs in this nation receive more credence than policy recommendations which point to the need for increased regulation of the marketing and advertising of alcoholic beverages or mandated health and safety warning labels as components of a program to reduce alcohol problems.

Some of the reasons for double policy standards for illegal drugs versus alcohol are obvious. There is no cocaine lobby in the Senate gallery when the Senate debates the war on drugs. The pervasive power of the alcohol beverage lobby is a fact of political life. Alcohol is undoubtedly the drug of choice of the United States Congress. It is simply easier for Senators and Congressman to discuss the drug usage of other Americans. But there are fundamental institutional problems as well which frustrate any concerted attempt to forge a comprehensive national policy to reduce alcoholism and alcohol-related problems.

The history of federal involvement in alcohol is a history of fragmentation, non-communication and agencies working at cross-purposes. Although NIAAA represents the heart of the federal alcohol effort, NIAAA lacks any authority over regulations or federal policy of any kind governing beverage alcohol. Despite NIAAA's placement within the Public Health Service, there has been complete resistance within both the executive and legislative branches of government to utilizing the public health model to address alcoholism and alcohol-related problems. Within the public health model, disease prevention strategies must address the agent, the host and the environment. It has taken us decades as a nation to resist blaming the host -- the individual with alcoholism for his or her condition. But our obsession with the host continues as we focus our attention almost exclusively on treating the illness, with little willingness to take necessary steps to reduce the size of the problem. And what is the legacy of this limited view? During debate of the Hughes Act in 1969, estimates of the cost of alcoholism and related problems to the nation were pegged at $12 billion. Today, the cost of these problems is slated to be $120 billion a year.

And how have federal institutions responded? While NIAAA funded treatment programs in the past and today funds research to identify a genetic predisposition to alcoholism, the Bureau of Alcohol, Tobacco and Firearms (BATF) continues to promulgate regulations in the economic interests of the alcohol beverage industry with little regard for the impact of any policy on the public health. The Food and Drug Administration (FDA) diligently monitors the dissemination of legal drugs to physicians and consumers while the nation's favorite legal drug, alcohol, continues to be exempt from any consumer information or protection initiatives implemented over the last two decades. The Federal Trade Commission (FTC) has repeatedly refused to investigate or further regulate alcohol advertising. This is while increasing numbers of ads promote alcohol as a beverage positively associated with health, sex, athletics, risky activities, economic and social success. These ads and other marketing strategies are the single greatest source of alcohol education for American children, but how frequently have we looked at them in the context of prevention and education programming?

Institutional problems call for institutional remedies. Government-wide concern over illicit drugs has resulted in increased coordination among federal agencies addressing this problem through the Interagency Board on Drug Abuse and the leadership of the White House Office on Drug Policy. No comparable coordinating body exists in the alcohol area. An HHS departmental workgroup in a report entitled "Options for the Organization of the Alcohol, Drug Abuse and Mental Health Administration" identified the absence of a coordinating mechanism for alcohol activities, indicating that "NIAAA's role in the Federal coordination of alcohol activities has not been articulated." NIAAA's 15 years of experience monitoring, assessing and defining the nature and extent of alcoholism and alcohol-related problems in the nation should inform all federal alcohol policy. Congress should delegate increased authority over alcohol regulation to the Department of Health and Human Services, either through the already existing regulatory mechanism of the Food and Drug Administration or through the creation of a new regulatory mechanism within NIAAA. Let BATF continue to collect alcohol excise taxes and to regulate industry activities which relate to basic commerce concerns. But industry activities which clearly affect the public health including pricing, promotion, approval for the manufacture of new products and advertising must be placed under an agency with expertise and a commitment to the impact of alcohol on health and safety. We cannot afford to continue to pretend that the proliferation of new products, deterioration in pricing, and ingenious new marketing strategies aimed at high-risk populations have no relation to the nature and extent of alcohol problems in the nation. Nor can we continue to deny a reality which is axiomatic in the public health community and in the prevention literature -- that there is a direct correlation between the amount of alcohol consumed and the level and severity of alcohol problems, including alcoholism. In a culture where nearly 36% of individuals do not drink and where 10% of the drinkers consume nearly 60% of the alcohol sold, consumption levels represent a critical barometer for alcohol problems.

While 1986 marked the beginning of a national strategy to address drug abuse, 1987 was the year for a national plan to reduce homelessness in America. It is telling that the earliest federal attention focused on alcoholics was inspired by public concern about the large numbers of public inebriates on the streets and in the jails of our communities. Detoxification and long-term treatment resources developed in conjunction with the federal call for the decriminalization of public drunkenness have been curtailed or eliminated in the eighties. This has been a direct result of federal retrenchment and the creation of alcoholism services for more politically desirable populations. As a consequence, many states have repealed decriminalization laws.

Public inebriates are visible in large numbers on the streets of our cities, and even the so called "new homeless", which include women and their children, present a very high rate of alcohol problems. Despite these realities, initial drafts of comprehensive homeless legislation in both the House and the Senate contained only a requirement in funding for the Health Resource Services Administration (HRSA) that substance abuse services be offered with no other provisions for this population. Concerted advocacy efforts ultimately produced $9.2 million in demonstration programs for alcoholic and other drug-dependent homeless individuals. A great deal more must be done if alcoholism's contribution to homelessness in America is to be adequately recognized and effectively addressed.

It is disturbing that a nation so up in arms about the pervasiveness of alcohol and other drug problems is so negligent about ensuring access to treatment to individuals with alcohol and other drug problems. The Federal Employees Health Insurance Benefits program is another case in point. Blue Cross-Blue Shield which services 70% of federal enrolees offers only detoxification coverage for alcoholics and other drug dependent people, and outpatient services only in psychiatric settings. As the Administration implements its drug-testing program, it is shocking that little consideration has been given to the inadequate treatment coverage most federal employees now have. The inadequacy of federal attention to treatment needs is also evidenced by the minimal coverage for these illnesses offered by the federal Medicare and Medicaid programs. Coverage for alcoholism is only available in hospital programs, with no provisions for care in the widely available and frequently less expensive free-standing residential programs. Outpatient coverage, although recently enhanced by mental health advocates, is still inadequate to provide for a comprehensive regimen of outpatient care. These policies stand despite the proliferation of research evidence which suggests that if we don't treat alcoholism, we treat the myriad and expensive medical complications alcoholism engenders.

In the meantime, access to alcoholism treatment for millions of Americans who are gainfully employed is now threatened by federal health insurance proposals and existing plans which provide no coverage for alcoholism treatment. NIAAA has done ground-breaking work in this area through research studies demonstrating the efficacy and cost-effectiveness of insurance coverage for alcoholism treatment and the promulgation of model benefit plans. Nevertheless Senator Kennedy's S. 1265, now on the Senate calendar and the Waxman companion measure in the House contain no coverage for alcoholism or other drug treatment. Moreover, the enactment of this legislation would preempt state mandated coverage for alcoholism treatment now present in a majority of states. It would also preclude any enhancement of the minimum benefit package through state legislative action. The enactment of this legislation without a provision for alcoholism and other drug treatment would set the field back decades and leave millions of Americans and their families without coverage for these treatable but life threatening illnesses.

Despite new funding and enhanced national attention to alcohol and other drug problems, federal policy clearly is not where it should be and in many instances the threat of losing ground is very real. Marty Mann, NCA's founder, identified the key mission of NCA in 1944 as a struggle against stigma. In 1988, stigma is very much with us, in the highest courts of the land and in the halls of Congress. On April 20, the U.S. Supreme

Court ruled that the Veterans Administration can deny certain benefits to alcoholic veterans on the grounds that their disability is a result of "willful misconduct." On April 27, the House Judiciary Committee narrowly defeated amendments to the Fair Housing Act which would have excluded alcoholics and alcohol abusers from anti-discrimination protections extended to disabled persons under the bill. An amendment excluding current drug abusers from protections was adopted and further weakening amendments are anticipated on the House floor. The recent debate on the Civil Rights Restoration Act was riddled with comments about a law which would force the hiring of alcoholics, drug addicts and other so-called undesirables. As an advocacy organization for alcoholic and drug dependent people and their families, NCA views the tenor of moralization and stigmatization as alarming. The civil rights of alcoholics and drug addicts are in jeopardy and the negation of these rights carries with it a real and formidable barrier to recovery for millions of Americans still suffering from these deadly illnesses.

If there is a single common denominator to all of these issues it is the crying need for federal leadership, particularly Congressional leadership. A former NCA official characterized the current state of affairs by saying that the federal government has taken the first step in Alcoholics Anonymous -- they have admitted they are powerless over alcohol. In 1984, we lost the Senate Alcoholism and Drug Abuse Subcommittee and this subcommittee was combined with a subcommittee addressing children and family issues. In the last two years, we have had only one Senate hearing on our issues, a subcommittee hearing on children of alcoholics and drug abusers. In the latest reauthorization cycle still underway, there were no hearings in the Senate on NIAAA, NIDA or the ADMS block grant although sweeping changes are proposed in both the Senate and House legislation. We cannot begin to compete with mental health or drug abuse interests for funding, because we simply have not had the advocates on Capitol Hill.

This series of hearings under the leadership of this Committee are the single most concerted attempt to review alcohol policy that our constituency has seen over the last eight years. Comprehensive anti-drug legislation is again on Congress's agenda with new opportunities to ensure the inclusion of policy and funding measures to address alcoholism and alcohol-related problems. This Committee has an important opportunity to take the testimony offered in these hearings and move to re-focus the nation's agenda toward a comprehensive and humane plan to reduce alcoholism and alcohol-related problems. I am convinced we have the knowledge today to make major strides in reducing the toll of the devastating illness of alcoholism in our lifetime. The National Council on Alcoholism stands ready to work in concert with you to meet this challenge.

Summary of Testimony to U.S.Senate Committee on Governmental Affairs

May 26, 1988

Witness: Donald W. Goodwin, M.D.
Professor and Chairman
Department of Psychiatry
University of Kansas School of Medicine
Kansas City, Kansas

Alcoholism runs in families. This has been known for centuries. Recent studies indicate that about 20-25% of sons of alcoholics become alcoholic and about 5% of daughters. These rates are approximately five-fold higher than the rates for alcoholism in the general population.

This does not mean that alcoholism is inherited. Many behaviors run in families that are clearly not inherited, such as speaking languages. Other familial traits are definitely inherited, such as blue eyes. Concerning alcoholism, is the transmission of the condition from generation to generation controlled mainly by environment or genes?

Attempts to resolve the "nature-nurture" problem in alcoholism has been of recent origin. The strongest evidence that alcoholism is influenced by heredity has come from adoption studies. Biological and environmental influences can be partly separated by studying individuals who were adopted in early infancy and raised by non-biologically-related adoptive parents. Three adoption studies were conducted in the 1970s and they provide the strongest evidence available to date supporting a genetic predisposition to alcoholism.

The studies were conducted in three places: Denmark, Sweden and Iowa. The U.S.-supported Danish studies found that sons of alcoholics were about four times more likely to be alcoholic than were sons of nonalcoholics even when both groups were adopted in early infancy and raised by moderate drinking adoptive parents. The findings for the daughters were more equivocal, with high rates of alcohol problems in both daughters of alcoholics and daughters of controls. The Swedish study produced essentially the same results, as did the Iowa study. However, based on the Iowa study, daughters of alcoholics also experience high rates of alcoholism when adopted and raised by moderate-drinking parents. The evidence for the daughters is still less conclusive than for the sons.

The adoption studies had remarkably similar findings in another regard: the fact that a person had a biological parent who was alcoholic increased the person's risk of becoming alcoholic but was not associated with increased risk of other psychiatric illnesses, such as anxiety disorders, depression and criminality. There may be one exception: sons of alcoholics may have a higher incidence of hyperactivity and conduct disorder in childhood.

While there are some contradictions in the adoption findings, the remarkable thing is their consistency. They show that alcoholism runs in families even when the children are separated from the alcoholic biological parents and raised by nonalcoholic adoptive parents. Moreover they do not show much, if any, overlap between alcoholism in the parents and other psychiatric disturbances in the offspring raised by adoptive families. This suggests that a genetic, or at least constitutional, vulnerability to alcoholism exists in families that is specific for alcoholism and does not increase the risk for other psychiatric illnesses.

The overall consistency of the adoption studies has encouraged researchers to explore mechanisms that might explain the increased vulnerability to alcoholism in individuals with a family history of alcoholism. One problem in isolating the vulnerability is that alcoholics, by definition, drink more alcohol than nonalcoholics and differences found between the two groups might be a consequence of drinking rather than a possible cause. One way to circumvent this is to study sons of alcoholics at high risk for becoming alcoholic before they begin heavy drinking.

Such studies are in progress in at least a dozen centers and some of their findings will be discussed by other witnesses. These so-called "high risk" studies are in fact longitudinal studies: identifying "markers" or predictors of future alcoholism depends on follow-up of subjects into the years of risk for alcoholism. This requires long-term funding. Only with follow-up, however, can individuals who develop alcohol problems be matched with predrinking variables that not only enhances prediction of whom becomes alcoholic but advances the search for causal factors in alcoholism.

I want to emphasize that each of these studies should be funded for follow-up. Without follow-up, the original investment will have been largely wasted. This is not to deny that some interesting differences have been found between groups of subjects, comparing sons of alcoholics to sons of nonalcoholics, but group differences are much less useful than individual correlations. These correlations can only be made after individuals develop drinking problems and this is why follow-up studies are essential.

(There is a long history in medicine and social sciences of longitudinal studies being launched at considerable expense and then forgotten as investigators lose interest or funding becomes unavailable. This is extremely wasteful of resources and when a longitudinal study is initiated provisions should be made, whenever possible, for completion of the study, even if the study lasts 20 years or longer.)

Meanwhile, important information about the cause or causes of alcoholism can be obtained without a long waiting period. Alcohol, for example, produces morphine-like substances in the brain and possibly individuals susceptible to alcoholism may produce these substances in excess amounts, thereby increasing the chance of addiction. There are various ways this possibility can be explored but so far they have been explored very little.

Other theories exploring a biological susceptibility to alcohol abuse involve various chemical transmitters in the brain such as serotonin and the endorphins. There is some evidence that acute intoxication increases serotonin activity in the brain followed by subnormal activity. New drugs on the market specifically enhance serotonin activity and there are preliminary data that these drugs modify drinking behavior in animals and humans. Studies of such chemical agents may produce emperical treatments for drug craving and withdrawal before the nature of craving and withdrawal are known. These studies should be pursued.

In closing, let me point out that not everyone who is alcoholic has alcoholism in the family. This has led to speculation that alcoholism can be divided into two types: familial and nonfamilial. Studies comparing alcoholics with a family history of alcoholism and those without such a family history have been conducted and differences found. The main difference has been a consistent finding that "familial" alcoholics develop their alcoholism at an earlier age than nonfamilial alcoholics and apparently have a more severe form of the illness.

Since the terms "Type 1" and "Type 2" alcoholism may be introduced at these hearings, I would like to state my provisional opinion that the two types overlap greatly with the concepts of familial and nonfamilial alcoholism. By the way, the idea that alcoholism has two forms - familial and nonfamilial - is an old one, dating back at least to the 19th century. The familial-nonfamilial comparisons should be further explored. If authentic, differences will ultimately reveal differences in causation, and only when causation is better understood will there evolve effective means of prevention and treatment.

GENETICS OF ALCOHOLISM

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Introduction

Alcoholism runs in families. This has been known for centuries. Only in recent years, however, has evidence emerged that alcoholism might be influenced by genetic factors. The principle evidence for a genetic factor comes from adoption studies that began in the mid-1970s and are still continuing. From the adoption studies it became clear that not only did alcoholism run in families but the transmission of alcoholism from parents to children occurred even when the children were raised by unrelated adoptive parents who were not heavy drinkers. The adoption work encouraged a large number of investigators to search for biological markers of alcoholism, mainly by studying children of alcoholics before they had begun drinking. Also, laboratory scientists increased their efforts to identify possible biochemical mechanisms explaining the transmission of alcoholism from parent to child. The genetic story continues to unfold; this review discusses some recent developments.

Adoption Studies

Adoption studies of alcoholism first began appearing in publications in the mid-1970s. They originated from three countries: Denmark, Sweden and the State of Iowa in the US. All came to remarkably similar findings: children of alcoholics had a high rate of alcoholism even when they were not raised by their alcoholic biological parents but were raised by adoptive parents whose drinking was light or moderate. (These studies are reviewed by Goodwin, Is Alcoholism Hereditary? Ballantine, 1988.) At first the findings applied mainly to sons of alcoholics but later the Iowa studies indicated similar increased risk in the adopted daughters of alcoholics. The first reports suggested that alcoholism in the biological parents predicted only alcohol problems in the adopted-away offspring and was not associated with other forms of substance abuse or other psychiatric illnesses. A recent study by Cadoret et al (1) reported that a biologic background of alcohol problems did predict increased drug abuse in adoptees, so the issue is not completely resolved.

Also, while the Danish studies suggested that environmental factors were relatively unimportant in the development of alcoholism, recent data from a Cadoret et al study (2) indicated that alcohol-related problems in the adoptive family also predicted increased alcohol abuse. Beardslee, Son and Vaillant (3), in a 40-year follow-up of working class families, found that exposure to alcoholism in the family environment and family history of alcoholism independently contributed to the later development of alcoholism.

Cloninger et al (Arch Gen Psychiat 1981,38,861-868) proposed that two types of alcoholism existed. Type I was relatively mild, occurred in both sexes, and was influenced by environmental factors (e.g., low economic status). Type II consisting only of men, involved a more severe family history of alcoholism and was manifested by antisocial features but was not influenced by social or environmental influences. The two-type hypothesis has attracted a good deal of attention but has just recently been the subject of replicative studies. Nordström and Berglund (4) found that Type II alcoholics, like their biological parents, represented a more severe group with a lower frequency of abstinence and social drinking and a tendency to benders when compared to Type I alcoholics.(Some have questioned whether the Type I alcoholic would really be considered alcoholic by usual clinical criteria.)

Another typology that has become popular recently involves dividing alcoholics into familial and nonfamilial alcoholics. Although alcoholism runs in families, it does not run in all families and there are many alcoholics who do not have a family history of alcoholism. By comparing alcoholics with a positive family history with those having a negative family history, interesting findings have been reported. These include an earlier age of onset and a more severe form of alcoholism in the familial alcoholics (5). Penick et al (6) recently confirmed the relationship of familial alcoholism to early onset and greater severity but also found that familial alcoholics had a increased lifetime prevalence of additional psychiatric disorders and a greater diversity of psychiatric disturbance among biological relatives. There is some controversy about the latter finding inasmuch as the children of alcoholics described in the adoption studies (by definition "familial") did not show an increased prevalence of additional psychiatric disorders. Penick suggests that assortative mating might explain the differences between familial and nonfamilial alcoholics. Possibly her population of Veterans Administration patients differed in important regards from the subjects in the adoption studies. In any case, whether familial alcoholism involves a greater or lesser risk of additional psychiatric disorders remains unresolved.

There are certain similarities between Cloninger's Type I and Type II alcoholics and familial and nonfamilial alcoholics. For example, both the Type II alcoholic and familial alcoholic have an early onset and involve a serious form of alcoholism (more so than occurs in nonfamilial alcoholism). Cloninger's proposal that heavy-drinking parents and low occupational status have additive effects in increasing alcoholism in the offspring has been supported by a recent study by Parker and Harford (7). Another environmental factor that might distinguish Type I and Type II alcoholism (or familial versus nonfamilial alcoholism) may involve maltreatment of children by the alcoholic parents. Alcoholics are overrepresented in the population of child abusers (8), and child abuse may occur more often in familial alcoholics than in nonfamilial alcoholics, although this needs confirmation.

Marker Studies

Evidence that alcoholism is genetically influenced has led to a search for biological markers of a predisposition toward alcoholism. Primarily this search has consisted of comparing children of alcoholics (a high risk group) with children of nonalcoholics (a low risk group). These studies are conducted before the subjects have had extensive exposure to alcohol and therefore differences between the two groups could be interpreted as possibly predispositional rather than a consequence of excessive drinking.

Schuckit (9), who pioneered so-called high risk studies, reviewed the major findings in this line of research. He focuses primarily on electrophysiological markers and cognitive and psychomotor markers.

Two types of brain waves have been identified as potential markers associated with a predisposition toward alcoholism. The first, event-related potentials (ERPs), are computer averaged brain waves measuring electrophysiological brain reactions to stimuli. One part of the ERP, a positive wave observed at 300 milliseconds after a stimulus (the P300), occurs in normal individuals after experiencing an anticipated rare event. The P300 reflects an individual's ability to selectively attend to an anticipated stimulus. Alcoholics have been reported to have a flattened amplitude of P300. In two laboratories (10) preadolescent sons of alcoholics also had decreased size of the P300 wave. However, a third laboratory (11) failed to document a relationship between family history and P300, leaving the importance of the finding still unresolved.

Schuckit (9) also cited studies reporting that alcoholics demonstrate less alpha activity on the EEG than nonalcoholics. Moreover, sons of alcoholics show increased alpha activity after an alcohol challenge. Schuckit notes that it is possible that alcohol has "different reinforcing properties in individuals at high risk for alcoholism, perhaps correcting for a lower level of alpha activity and, thus, producing more feelings of relaxation."

Schuckit (9) further reviews studies reporting that high risk subjects demonstrate decreased verbal IQs, decreased auditory word span performance, have lower level of reading comprehension and make more errors on the Categories test of the Halstead-Reitan Battery, as well as have problems with constructional praxis and abstract problem solving.

One consistent finding in Schuckit's own studies has been a lower intensity of reaction to alcohol in the sons of alcoholics. They seem to have an innate tolerance to alcohol. They become less intoxicated when drinking and have lower cortisol and prolactin levels in response to a high-dose ethanol challenge (12).

Other differences reported between sons of alcoholics and controls include:

(1) High-risk subjects are more cardiovascularly reactive to a shock stressor than a moderate-risk group when sober(13). Alcohol consumption causes a "dramatic" reduction in the degree of reactivity in the high-risk group and leads to increased reactivity in the moderate-risk group. Conclusion: Prealcoholic sons of alcoholics are more sensitive to stress than controls and alcohol reduces this sensitivity markedly.

(2) This potentially reinforc~~ed~~ing effect of alcohol was demonstrated in another study (14) where alcohol's capacity to attenuate physiological responses to stress was more pronounced in high-risk subjects than in low-risk subjects.

(3) Sons of alcoholics had higher based thyrotropin levels than sons of nonalcoholics (15). The difference was

not found between daughters. This is the first report of a male-limited neuroendocrine difference between children of alcoholics and control children.

Perceptual field dependency, characterized by an inability to disciminate visually a stimulus from a distracting background, has been reported in alcoholics. Tarter et al (16), using the rod and frame test, found no differences in field dependency between sons of alcoholics and sons of depressed fathers and control fathers.

Schuckit (9) reviews the literature regarding another type of marker: the possible role of hyperactivity in childhood as a predictor of alcoholism. He notes that several studies indicate that alcoholics are more likely to report childhood hyperactivity than nonalcoholics, but other studies (including one in his own laboratory) failed to confirm the association. In another paper (17), however, he notes that alcoholics' sons report more evidence of a hot temper and short attention span than sons of nonalcoholics. He found no association between alcoholism in the family and attention deficit disorder (ADD), residual type, in adulthood. Two studies (18), however, identified a "subtype" of alcoholism in which about one-third of young male alcoholic patients displayed symptoms of ADD, beginning in childhood and continuing into adulthood as a residual type disorder. The relationship between alcoholism and ADD still is poorly understood.

A marker of nonalcoholism has been much studied in recent years. This is the so-called Oriental flush. About half of Orientals develop a cutaneous flush and experience unpleasant subjective feelings when drinking a small amount of alcohol. The reaction is believed to be genetically determined. Recent evidence for this (19) was produced by Japanese investigators reporting that subjects who experience a flush after drinking were deficient in the enzyme that most efficiently metabolizes acetaldehyde. The flushing is believed caused by an increase of acetaldehyde. Orientals with the deficient enzyme experience unpleasant reactions to alcohol from early infancy. This discourages alcohol consumption and may largely account for the generally low rate of alcoholism in the Orient.

What is Inherited?

To the extent that a tendency toward alcoholism is inherited, it is biochemical in origin. The chemistry of the brain is still largely a mystery, and even less is known about the effects of alcohol on brain chemistry. Nevertheless, several hypotheses are being pursued and will be discussed briefly.

First, persons with a genetic propensity for alcoholism may be deficient in certain forms of biochemical activity required for optimal well-being. These persons, given available alcohol, a suitable culture, and an absence of countervailing traits, might discover that alcohol temporarily corrects this hypothetical deficiency, producing an intensity of mood change foreign to those without the deficiency. The model then requires that alcohol would have a biphasic effect, causing subsequent underactivity of the reward system.

There is some evidence that alcohol has a biphasic effect on serotonin metabolism that might then correspond to the deficiency model. Like reserpine, alcohol appears to increase serotonergic activity briefly (during acute intoxication) and then subsequently reduces serotonin activity to subnormal levels. The "deficit" person then would have two reasons for drinking. He first would drink to correct the deficiency and then continue to drink to correct for an even greater deficiency resulting from the biphasic effect of alcohol on serotonin. Biochemically, this might explain the "addictive cycle" in which a person initially drinks to feel good and then later drinks to stop feeling bad from the substance that originally made him feel good.

The serotonin deficiency hypothesis has gained support from several recent studies, some of which are reviewed by Roy et al (20). The review concludes that a sizable subgroup of alcoholics have a reduced central serotonin turnover. The "reason" for drinking may be to correct this presumed deficiency.

A series of studies by Boismare et al (21) of animals and human subjects strongly supports the serotonin deficiency hypothesis. They found that alcohol-drinking rats showed an increased serotonin uptake compared to non-alcohol-drinking rats, studying striatal synaptasomes. They also found that antidepressants that inhibit serotonin uptake decrease alcohol consumption by alcohol-drinking rats. Pursuing this work with humans, they reported that platelet uptake of serotonin was increased in alcoholics and former alcoholics compared to controls. The phenomenon could be congenital or induced by previous excessive intake of alcohol. If congenital, it would represent a true biological marker for a predisposition to alcoholism.

Another deficiency hypothesis involves endorphins. The theory holds that alcoholics have decreased levels of endorphins in the nervous system and that alcohol compensates for the deficiency. A recent paper by Bourhis et al (22) is consistent with the hypothesis, showing increased levels of beta-endorphins in intoxicated alcoholics. Other recent works (23) suggest that opioid receptors influence the development of ethanol dependence. Opioid antagonists, for example, block the ethanol withdrawal syndrome in rats. The endorphin theory has less supporting data than the serotonin hypothesis.

A third deficiency hypothesis involves catecholamines. The depletion of central catecholamines by inhibitors of tyrosine hydroxylase or dopamine-beta-hydroxylase reduces voluntary intake of ethanol in rodents (24). Furthermore, the activity of tyrosine hydroxylase is higher in brains of alcohol-preferring rats than in those of alcohol-non-preferring rats (24). There is still little work in humans to support the hypothesis. A good review of neurotransmitters, cell receptors and ethanol was recently published by Bannister and Losowsky (25) .

In addition to deficiency theories, there is also an "overproduction" theory of alcoholism. This theory holds that a genetic propensity to alcoholism involves the overproduction of substances that in some way facilitate addiction. For example, alcohol produces minute amounts of morphine-like compounds in the brain. One study (25) found these compounds in the spinal fluid of alcoholics in greater quantities after alcohol ingestion than in nonalcoholics. Rats and monkeys drink increased amounts of alcohol when these substances are injected into the brain (25). Thus, the possibility exists that the genetic defect in alcoholism involves a reduced capacity of the brain to oxidize aldehydes, resulting in the overproduction of morphine-like alkaloid compounds (aldehyde condensation products), which may facilitate alcohol addiction.

Some laboratories are planning DNA sequencing studies in an attempt to locate a specific gene or genes for alcoholism (the ultimate marker). So far these studies have not produced data. A recent report from Japan (26) found an elevation of chromosomal aberrations in the lymphocytes of alcoholics compared with those of nonalcoholics. However, when the alcoholics stopped drinking, there was a spontaneous reduction of the aberrations, suggesting they were caused by alcohol and reversible.

Annotated references and recommended reading

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* Selected for review

** Of outstanding interest

1. Cadoret RJ, Troughton E, O'Gorman TW, and Heywood E: An adoption study of genetic and environmental factors in drug abuse. Arch Gen Psychiatry 1986,43:1131-1136.

* A biologic background of alcohol problems predicted increased drug abuse in adoptees, contradicting the results of the Danish adoption study.

2. Cadoret RJ, Troughton E and O'Gorman TW. Genetic and environmental factors in alcohol abuse and antisocial

personality. Journal of Studies on Alcohol 1987,48:1-8.

* Alcoholism in the biologic parents predicts alcoholism in their adopted children. Alcoholism in the adoptive family also predicts increased adoptee alcohol abuse.

3. Beardslee WR, Son L and Vaillant GE: Exposure to parental alcoholism during childhood and outcome in adulthood: A prospective longitudinal study. British Journal of Psychiatry 1986,149:584-591.

* Another study showing the importance of exposure to alcoholism in the family environment.

4. Nordström G and Berglund M: Type 1 and Type 2 alcoholics (Cloninger & Bohman) have different patterns of successful long-term adjustment. British Journal of Addiction 1987,82:761-769.

** More bender drinking and less abstinence and social drinking in Type 2 alcoholics (a la Cloninger).

5. Haver B. Female alcoholics V: The relationship between family history of alcoholism and outcome 3-10 years after treatment. Acta Psychiatr Scand 1987,76:21-27.

* More evidence that familial alcoholism has an early onset.

6. Penick EC, Powell BJ, Bingham SF, Liskow BI, Miller NS and Read MR: A comparative study of familial alcoholism. Journal of Studies on Alcohol 1987,48:136-146.

** Again, familial alcoholics have an earlier onset and a more severe type of alcoholism, plus (unexpectedly) more psychiatric disorders than nonfamilial alcoholics. Assortative mating may be the explanation.

7. Parker DA and Harford TC: Alcohol-related problems of children of heavy-drinking parents. Journal of Studies on Alcohol 1987,48:265-268.

** Heavy-drinking parents and low occupational status places children at elevated risk for alcohol-related problems (Cloninger's Type 1 alcoholism). Note that the term is "heavy drinking" rather than "alcoholic."

8. Famularo R, Stone K, Barnum R and Wharton R: Alcoholism and severe child maltreatment. Amer J Orthopsychiat 1986,56:481-485.

* More evidence that alcoholics abuse children.

9. Schuckit MA: Biological markers in alcoholism. Prog Neuro-Psychopharmacol & Biol Psychiat 1986,10:191-199.
** Schuckit is the father of high-risk studies in alcoholism and this excellent review discusses some of his own work and that of others.

10. O'Connor W, Hesselbrock V and Tasman A: Correlates of increased risk for alcoholism in young men. Prog Neuro-Psychopharmacol & Biol Psychiat 1986,10:211-218.
** Confirms earlier studies reporting that sons of alcoholics produce smaller amplitudes of the P300 wave than sons of nonalcoholic fathers.

11. Polich J and Bloom FE: P300 and alcohol consumption in normals and individuals at risk for alcoholism. A preliminary report. Prog Neuro-Psychopharmacol & Biol Psychiat 1986,10:201-210.
** A contradiction in the P300 literature: this excellent group at the Scripps Clinic did not find an association of P300 amplitude and parental alcoholism.

12. Schuckit MA, Gold E and Risch C: Serum prolactin levels in sons of alcoholics and control subjects. Am J

Psychiatry 1987,144:854-859.

* Schuckit finds that sons of alcoholics underrespond to alcohol. They even have lower prolactin levels in response to ethanol challenge.

13. Finn PR and Pihl RO: Men at high risk for alcoholism: The effect of alcohol on cardiovascular response to unavoidable shock. Journal of Abnormal Psychology 1987,96:230-236.

** Maybe this is why some people become alcoholics and most do not: Sons of alcoholics have increased heart rate when subjected to shock while sober. While drunk their heart rate is dramatically reduced. Conclusion; a predisposing factor in alcoholism is a sensitivity to stress that is relieved by drinking. An old theory that still needs more proof.

14. Levenson RW, Oyama ON and Meek PS: Greater reinforcement from alcohol for those at risk: Parental risk, personality risk and sex. Journal of Abnormal Psychology 1987,96:242-253.

** Another study showing that alcohol is more "reinforcing" for sons of alcoholics because it attenuates physiological responses to stress to a greater degree than in sons of nonalcoholics.

15. Moss HB, Guthrie S and Linnoila M: Enhanced thyrotropin response to thyrotropin releasing hormone in boys at risk for development of alcoholism. Arch Gen Psychiatry 1986,43:1137-1142.

- Sons of alcoholics have higher basal thyrotropin levels than sons of nonalcoholics. Does this suggest that sons of alcoholics suffer from subclinical hyperthyroidism and maybe this contributes to their drinking? Possible but not proven.

16. Tarter RE, Jacob T, Hill S, Hegedus AM and Carra J: Perceptual field dependency: Predisposing trait or consequence of alcoholism? Journal of Studies on Alcohol 1986,47:498-499.

- Sons of alcoholics have no more field dependence than controls.

17. Schuckit MA, Sweeney S and Huey L: Hyperactivity and the risk for alcoholism 1987, J Clin Psychiatry,48:275-277,

- Reports that alcoholism is associated with attention deficit disorder received no confirmation in this study.

18. Morton AM, Fiscella RA, O'Connor K, Jackson M and Slone DG: Revised criteria for detecting alcoholic patients with attention deficit disorder, residual type. The Journal of Nervous and Mental Disease 1987,175:371-372.

* This is one of two reports that hospitalized alcoholics have a high rate of attention deficit disorder, residual type. The hyperactivity-alcoholism link is still viable.

19. Ohmori T, Koyama T, Chen CC, Yeh EK, Reyes BV and Yamashita I: The role of aldehyde dehydrogenase isozyme variance in alcohol sensitivity, drinking habits formation and the development of alcoholism in Japan, Taiwan and the Phillipines. Prog Neuro-Psychopharmacol & Biol Psychiat 1986,10:229-235.

** Many Orientals flush when they drink alcohol. It is now known why: they lack the efficient form of aldehyde dehydrogenase.

20. Roy A, Virkkunen M and Linnoila M: Reduced central serotonin turnover in a subgroup of alcoholics? Prog Neuro-Psychopharmacol & Biol Psychiat 1987,11:173-177.

** A good review suggesting that some alcoholics have a serotonin deficiency.

21. Boismare F, Lhuintre JP, Daoust M, Moore N, Saligaut C and Hillemand B: Platelet affinity for serotonin is increased in alcoholics and former alcoholics: A biological marker for dependence? Alcohol & Alcoholism 1987,22:155-159.

** A study suggesting why some alcoholics may have a serotonin deficiency. Their cells have an increased affinity for serotonin and thus less of the neurotransmitter is available at the synapse.

22. Barret L, Bourhis F, Buffet H, Danel V and Debru JL: Determination of β-endorphin in alcoholic patients in the acute stage of intoxication: relation with naloxone therapy. Drug and Alcohol Dependence 1987,19:71-78.

* Naloxone-treated subjects (both alcoholics and controls) while intoxicated with alcohol resulted in increased levels of beta-endorphin. More support for the role of the opioid system in alcohol metabolism.

23. Kotlińska J and Langwiński R: Does the blockade of opioid receptors influence the development of ethanol

dependence? Alcohol & Alcoholism 1987,22:117-119.

* Opiate antagonists changes the manifestations of alcohol withdrawal in rats, more evidence of a biochemical link between the action of ethanol and opiates at the level of opioid receptors.

24. Pispa JP, Huttunen MO, Sarviharju M and Ylikahri R: Enzymes of catecholamine metabolism in the brains of rat strains differing in their preference for or tolerance of ethanol. Alcohol & Alcoholism 1986,21:181-184.

* Evidence in animals that depletion of central catecholamines reduces voluntary intake of ethanol.

25. Bannister P and Losowsky MS: Cell receptors and ethanol. Alcoholism: Clinical and Experimental Research 1986,10:50S-54S.

** An excellent review of neurotransmitters, cell receptors and ethanol.

26. Matsushima Y: Chromosomal aberrations in the lymphocytes of alcoholics and former alcoholics. Neuropsychobiology 1987,17:24-29.

* Alcoholics had chromosomal aberrations but they were reversible.

PREPARED STATEMENT OF BARBARA LEX, Ph.D., M.P.H

Mr. Chairman, members of the committee. My name is Barbara W. Lex. I am Assistant Professor of Psychiatry at the Harvard Medical School, Alcohol and Drug Abuse Research Center, McLean Hospital. I am pleased to be here today to present information about the status of research on alcohol effects on women. During the past seven years our research group has conducted multidisciplinary studies of substance abuse effects in women. For the past three years my work has compared sensitivity to alcohol effects in women with and without a family history of alcoholism. Before summarizing these findings, I will present some background information that will help to place this work in scientific context:

- Currently it is estimated that about 5% of women who drink become alcohol dependent, and about 25% of persons who receive treatment for alcohol-related problems are female. Information about causal association between alcohol dependence and predisposing factors in women is not as complete as we might wish. However, it is thought that some growth of drinking problems among women may reflect higher rates associated with changing social norms, but also may stem from diminished stigma associated with seeking help, improved recognition of alcohol problems, and expansion of treatment programs. For example, until a year ago judges in Massachusetts had no recourse but to order women in need of alcoholism and drug abuse treatment to a holding facility in the state prison for women. When an appropriate inpatient program for women requiring civil commitment for treatment opened in Boston, I evaluated the psychological and social characteristics of the first 20 women who were admitted. All 20 had multiple medical, reproductive, psychological, social, and economic problems, and 85% had a family history of alcoholism.
- No single characteristic explains or predicts alcohol problems, and no single study can examine all potentially relevant variables. It is currently believed that alcoholism in women, as in men, results from a combination of behavioral, biological, and sociocultural factors. These factors are so

interwoven in processes promoting and maintaining alcohol dependence that only since the 1970s has there been sufficient information to begin separating the potential influence of background factors from the consequences of excessive alcohol intake.

- The extent that psychosocial and biological factors differentially contribute to alcohol dependence is incompletely known and requires further systematic investigation. Since offspring of alcoholics are 3 to 4 times as likely as persons in the general population to develop alcoholism (20-30% versus 5-10%), a useful strategy for disentangling complex contributory factors is to study persons at high risk for alcohol abuse. Studies examining biological vulnerability to alcoholism need to be conducted before the onset of alcohol abuse produces physiological, cognitive, or emotional changes. However, our knowledge of specific characteristics in young women that might predict development of alcohol abuse later in their lives is not as well defined as information about parallel processes in men.
- Historically, alcohol problems in women received little systematic attention. It was believed that sociocultural factors protected women from alcohol abuse, that alcohol dependence was similar in men and women, or that alcoholic women were more deviant, psychologically disturbed, and difficult to study and treat. However, comparisons of family backgrounds show that alcoholic women are more likely than alcoholic men to have an alcoholic parent, especially fathers, and that prevalence of alcoholism in fathers of alcoholic women in treatment may be as high as 61%. More women than men who are treated for alcohol problems report that their mothers, siblings, or spouses also have alcohol problems.
- The impact of genetic and familial factors has been amply demonstrated in studies of alcohol problems in male adoptees in Scandinavian countries and in the United States. Nearly 4 times the rate of alcoholism was found in adopted-away

sons of two types of alcoholic biological parents than in sons of nonalcoholics. One study of female adoptees in Denmark included an insufficient number of alcoholic women for definite conclusions but suggested that alcoholism in women may have a partial genetic basis. A larger study in Sweden showed influence of paternal alcohol abuse on adopted-out daughters whose biological fathers had later onset of alcoholism, had incurred only minor legal infractions, and required minimal alcoholism treatment ("milieu-limited" or "Type 1" alcoholism). Lower socioeconomic status of adoptive parents and rearing in rural areas were environmental factors associated with alcohol abuse rates. However, women whose alcoholic fathers had severe early onset alcohol dependence associated with criminal behavior ("male-limited" or "Type 2" alcoholism) tended to be treated for many episodes of headaches, backaches, and abdominal distress. While hereditary factors might seem to contribute *less* to female alcoholism, paternal alcoholism nevertheless is potentially important for women--especially since a recent survey of social drinkers found that more women than men reported having alcoholic fathers. It is now recognized that age of onset, drinking patterns, and symptoms of alcohol dependence are different for alcoholic women and men. Drinking problems in women typically begin 4 to 8 years later than in men, but alcohol dependence develops more rapidly even though women drink less frequently, and consume less alcohol. Women report that before coming to treatment they had shorter drinking histories and fewer episodes of binges, continuous drinking, blackouts, morning drinking, and delirium tremens. Numerous studies associate women's alcohol abuse with gynecological dysfunction, early menopause, serious fetal defects, and more rapid development of life-threatening cardiovascular, gastrointestinal, and liver diseases.

- It is hypothesized that in daily life individuals use assessments of intoxication as cues to reduce or cease drinking. It is that believed young men with a family history of alcoholism are less affected by moderate doses of alcohol. If cues to reduce or stop drinking are indistinct, delayed, or lacking, then sons of alcoholic fathers may be predisposed to consume more alcohol, become more tolerant to alcohol effects, continue to consume alcohol, and thus accelerate the process of becoming alcohol dependent. Carefully conducted studies found that after 2 to 3 drinks, men with alcoholic fathers showed less overall body sway and rated themselves significantly less intoxicated than sons of controls, but had comparable alcohol metabolism rates and similar alcohol levels in their blood.

No comparable studies existed for women before we tested this hypothesis. We are currently studying healthy young women with and without alcoholic fathers. Women are carefully screened and matched for education, height, and weight, and they are selected because they drink no more than the equivalent of one beer per day. All women are studied during the same phase of the menstrual cycle. Findings from our early studies show that differential responses to alcohol effects can be identified in daughters of alcoholic fathers.

In our studies we used the a moderate dose of alcohol that had shown differential responses in studies of young men. The appropriate dose of alcohol for each woman is calculated according to her body weight, mixed with grapefruit juice, and placed in a special drinking container that gives off a strong alcohol taste and odor. Since studies of men showed that body sway is affected by sensitivity to alcohol (and other psychoactive drugs), we designed a special device to measure body sway and tested cognitive changes with a symbol matching test.

To date, our results show that levels of alcohol in blood, the rate of alcohol metabolism are comparable for women with and without alcoholic fathers. More importantly, like sons of alcoholic fathers, daughters of alcoholic fathers *sway less* after drinking alcohol. However, in contrast to findings for men after drinking the same amount of alcohol, intoxication scores for the comparison groups of women in our studies were similar and women's ratings of alcohol effects were lower. In addition, the daughters of alcoholic fathers performed better on a symbol matching test.

One possible explanation for apparent differences between the two series of studies might be that results reflect individual differences. However, various comparisons of individuals with and without a family history of alcoholism generally indicate some type of differential sensitivity to alcohol. Studies of differences in prepubertal and adolescent children of alcoholics and controls have suggested differences in central nervous system sensitivities, and some results are in the opposite direction of findings from young adults who have been social drinkers for several years. However, it is possible to reconcile seemingly disparate findings by hypothesizing that despite similar patterns of social drinking in young adulthood, greater sensitivity to the effects of alcohol may lead to greater tolerance for alcohol in persons who have a family history of alcoholism.

Differences between men and women may also affect results, but to the best of our knowledge studies of male-female differences as yet have not controlled for family history of alcoholism. Although our information is still limited, women may generally use different cues to monitor their level of intoxication. In comparison to men, young women have been reported to experience more anxiety associated with the social consequences of alcohol consumption. It is fair to say that women in general have different life experiences associated with alcohol, including possible self-medication with alcohol to relieve mood changes and physical discomfort associated with disorders of the reproductive system. Recent research seems to indicate that alcohol interferes with the balance of co-factors necessary for the metabolism of reproductive hormones, such as estradiol. Clearly, effects of female gender and family history of alcoholism on alcohol expectancies and intoxication ratings require investigation through carefully designed studies with sufficiently large samples of women who have clearly defined characteristics.

COMMENTARY

Further tests of effects of family history of alcoholism on sensitivity to alcohol in men and women will continue to require careful assessment by a number of physiological, subjective, and behavioral measures. One major concern emerges from this brief review and comparison of selected relevant findings. First, sample sizes that are small may yield results influenced by uncontrolled biases: larger samples generate more stable statistics. However, if findings are to be replicated, then sample selection must be guided by controlling for intervening variables. This means that it will be necessary to control for menstrual cycle effects in studies of women until there is evidence to the contrary. A significant trade-off factor, however, is that careful screening, matching, and controlling for possible biasing factors will inevitably prolong data collection until a sufficiently large sample can be studied. Accordingly, it is important to examine several factors in one study. Moreover, if equivalent variables and similar testing methods are used by a number of investigators then results can be compared more readily within a sufficiently large data base.

Further, numerous factors contribute to vulnerability conveyed by inheritance of alcoholism, including family environment and societal trends that favor or discourage increased consumption. At this juncture only 40% to 70% of sons of alcoholic fathers can be definitively classified by a profile of behavioral and biological markers. We will be able to develop an equivalent profile of markers for women as we continue to evaluate several dimensions of sensitivity to alcohol. Thus far our studies of effects of family history of alcoholism on women have confirmed body sway as one marker in women, and performance on more complex symbol-matching tasks may be an additional marker. In this way increased understanding will emerge as more data from testing additional variables become available. At this time we foresee that data continue to need interpretation using appropriate models of both psychosocial and biological contributions to the mechanisms of inheritance and transmission of vulnerability to alcohol abuse.

Sufficient resources are necessary for continued investigation of other factors involved in vulnerability to alcoholism, the relative contributions of genetic/familial and environmental factors to alcohol problems, and appropriate models for education, intervention, and treatment. Within the last decade concerns about children affected by parental alcoholism have come to public attention. Special research and policy task forces and commissions have convened to examine the magnitude of their problems, and some estimate that about 28,000,000 Americans have at least one alcoholic parent. However, as yet there is no clear consensus among clinicians and scientists about the type, extent, or severity of possible impaired functioning in children of alcoholic parents, nor do we know what might constitute the most effective formats for intervention and treatment. One logical outcome of research on the effects of family history of alcoholism could be earlier diagnosis and earlier treatment for vulnerable persons. Since ethical considerations argue for an interrelationship between prevention and intervention programs, however, these topics must be addressed before beginning any broad educational campaign.

STATEMENT

on

ALCOHOLISM AND FAMILY HISTORY IN WOMEN

FOR PRESENTATION TO

SENATE GOVERNMENTAL AFFAIRS COMMITTEE:

by

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INTRODUCTION

Alcohol abuse by women in the United States is now recognized as an important but incompletely studied chronic health problem [5, 6, 12, 28, 46, 56, 77, 91]. Currently it is estimated that about 5% of women who drink alcohol become alcohol dependent [13, 94], but about 25% of persons who receive treatment for alcohol-related problems are female [11, 58]. Apparent growth of drinking problems among women may reflect higher consumption by women ages 35 to 49 associated with changing social norms which have affected women since the 1950s, but also may stem from improved recognition of problems associated with alcohol and lessened stigma associated with seeking help [2, 22, 46, 56, 94].

No single characteristic explains or predicts alcohol problems [15, 46, 52, 55, 53]. It is currently believed that alcoholism in women, as in men, results from a combination of behavioral, biological, and sociocultural factors [26, 33, 46, 53, 56, 59, 70]. These factors are so interwoven in processes promoting and maintaining alcohol dependence that only since the 1970s has there been sufficient information to begin separating the potential influence of background factors from the consequences of excessive alcohol intake [55].

Historically, alcohol problems in women received little systematic attention, largely because it was variously believed that sociocultural factors protected women from alcohol abuse, that alcohol dependence was similar in men and women, or that alcoholic women were more deviant, psychologically disturbed, and difficult to study and treat [3, 12, 14, 46, 56] . It is now recognized that age, drinking patterns, and symptoms of alcohol dependence are different for alcoholic women and men [10, 14, 16, 38, 46, 53, 64, 77]. Onset of drinking problems in women occurs 4 to 8 years later than in men [5, 42, 48, 71, 72], but alcohol dependent women drink less frequently and consume less alcohol [42, 72, 77], report fewer binges and continuous drinking [72, 77, 93], have fewer blackouts, morning drinking, and delirium tremens episodes [72, 88], and report that before coming to treatment they had shorter drinking histories [5, 20, 21, 48, 82]. Thus, alcohol problems can seem to be less severe for women. However, numerous reports associate alcohol abuse in women with serious gynecological and obstetric dysfunctions [27, 53, 54, 90] as well as with a process called "telescoping," that is, more rapid development of cardiovascular, gastrointestinal, and liver diseases [8, 35, 46, 94]. Effects of alcohol on the developing fetus also cannot be discounted [25, 46, 53].

Processes leading to these consequences are incompletely known. The relative contributions of psychosocial and biological factors to disease outcomes will not be identified without further systematic investigation [46, 53]. It is also important to acknowledge that women who have alcohol problems are at risk for polydrug use, including use of such dependency-producing substances as psychotropic prescription medications, marijuana, and cocaine [14, 46]. A combination of familial, genetic, and environmental factors also are important for intercurrent polydrug use [14].

One consistent finding is that offspring of alcoholics generally are 3 to 4 times as likely as persons in the general population to develop alcoholism (20-30% versus 5-10%) [10, 19, 29, 59, 86]. Accordingly, a useful strategy for disentangling the multiplicity of contributory factors is to examine specific factors in persons at high risk prior to the onset of alcohol abuse. Our knowledge of specific characteristics in young people that might predict development of alcohol abuse later in their lives is limited by small sample sizes and by biases in education and income levels in the few available longitudinal and follow-up studies of male and female drinking patterns [23, 24, 44]. Nevertheless, comparisons of family backgrounds of alcoholics report that women are more likely than men to have alcoholic parents, especially the father [5, 19, 30], that prevalence of alcoholism in fathers of alcoholic women in treatment is as high as 61% [18], and, that in contrast to alcoholic men in the same treatment populations, alcoholic women have higher rates of alcoholism than their parents, siblings, or spouses [48, 83]. Concordance between alcoholism and affective disorders in the same family also has been reported to be as high as 50% for the mothers and sisters of alcoholic women [96, 97].

The impact of genetic and familial factors has been amply demonstrated in studies of alcoholism problems in male adoptees in Scandinavian countries and in the United States [29, 31, 79]. Nearly 4 times the rate of alcoholism was found in adopted-away sons of Danish alcoholic biological parents than in sons of nonalcoholics [29]. In a similar study of Danish female adoptees [30] it was suggested that alcoholism in women may have a partial genetic basis, and in a larger study of Swedish women adoptees [10] the influence of paternal alcohol abuse was seen in the backgrounds of adopted daughters with biological fathers who became alcoholic later in life and incurred only minor legal infractions, that is, Type 1 alcoholics [15, 16]. History of foster care was similar for alcoholic and nonalcoholic women, but environmental factors such as lower socioeconomic status of adoptive parents and rearing in rural areas were found to affect alcohol abuse rates [cf. 69]. Thus, while hereditary factors might seem to contribute less to female alcoholism [2, 32], parental alcoholism nevertheless is potentially important for women [73], especially since more women than men in a recent U.S. survey sample reported having alcoholic fathers [60].

The high risk of alcoholism in offspring and siblings of alcoholics gives evidence favoring the importance of genetic factors, and argues strongly for investigation of potential biological vulnerability to development of alcoholism. Comparative studies conducted before the onset of alcohol abuse can occur [e.g., 7, 70, 80, 89] are preferable to comparisons of alcoholics with non-alcoholic controls because they avoid potential confounding imposed by effects of alcohol-induced physiological, cognitive, or emotional changes [7, 56, 80, 81, 89].

Young men with a family history of alcoholism are believed to be vulnerable to alcohol effects, that is, either less sensitive or reactive to the subjective and physiologic effects of moderate doses of alcohol. One series of studies [76, 78, 80, 81] identified possible markers for genetic contributions to alcoholism by comparing acute alcohol effects in healthy young men (aged 21 to 25 years) who had no histories of alcohol abuse, drug abuse, or psychological disturbance and positive (FHP) or negative (FHN) histories of alcoholism in first degree relatives. FHN and FHP men were matched for age, height-weight ratio and drinking history, and were randomly assigned to receive testing after they drank alcohol placebo, 0.56 g/kg alcohol, and 0.82 g/kg alcohol beverages. After a typical social dose (0.56 g/kg alcohol), FHP men had blood alcohol levels and clearance rates equivalent to FHN controls, but showed less overall body sway and rated themselves significantly less intoxicated [76, 81]. Findings from these and other studies of young men at high risk for alcoholism suggested that young FHP men in some way may be differentially sensitive to subjective effects of moderate (social) alcohol doses [76, 81]. It is hypothesized that in daily life individuals use assessments of intoxication as internal cues to reduce or cease drinking. [47, 76, 81]. If such cues are indistinct, delayed, or lacking in FHP drinkers, then FHP individuals may be predisposed to consume more alcohol, develop increased tolerance to alcohol effects, continue to consume more alcohol, and subsequently become alcohol dependent. No comparable study existed for young women before we tested this hypothesis.

ALCOHOLISM AND FAMILY HISTORY IN WOMEN

Findings from pilot studies in our laboratory show that certain differential sensitivities to alcohol observed in FHP men also can be identified in FHP women. We are currently studying healthy female volunteers ages 21 to 25 to compare alcohol effects on subjects with and without alcoholic fathers. Women are carefully screened and matched for menstrual cycle phase, age, education, height and weight, and they are selected because they drink no more than the equivalent of one beer per day.

In one pilot study we compared for 6 FHP and 6 FHN women who were carefully screened and matched for education, drinking history and height/weight ratio (range: weight, 49.7 to 80.0 kg; height: 153.0 to 176.5 cm) between the ages of 21 and 25 (X = 23.1 years). Subjects provided informed consent for paid participation and were examined by a physician. Only physically healthy women who had no history of drug or alcohol abuse, or other psychological disorders were selected. To avoid confounding by possible effects of fetal exposure associated with maternal alcoholism [47], only daughters of alcoholic fathers were selected for the FHP category, and women who reported no alcohol problems or other major psychological disorders in primary relatives (full siblings and biological parents) were designated FHN. Each subject was individually tested 6 to 10 days after the beginning of menstruation (during the follicular menstrual cycle phase) to avoid possible confounding of mood or metabolic effects of hormonal fluctuation across the menstrual cycle [34, 36, 41, 56, 63, 62].

METHODS

On Day 1 of our 2-day experiment subjects are further screened for major psychological disorders, assessed for possible differences on cognitive and performance tasks, and have 3 practice trials of the test battery to minimize the effects of learning. All subjects are instructed to refrain from alcoholic beverages on the day prior to the experiment, and no solid food, caffeinated beverages, or tobacco cigarettes could be consumed on Day 2 between midnight and 2 p.m., when the experiments end.

On Day 2 subjects came to the laboratory in the morning, two hours before beverage administration. Subjects are tested for recent alcohol or drug use. To establish baseline values performance tests are conducted at 90, 60, and 30 minutes prior to beverage administration. Subjects also complete a 23-item alcohol-effects expectancy scale [77] 30 minutes before receiving the experimental beverage. Scale items include emotional states (e.g., happiness, tension), physical symptoms (palpitations, weakness, sleepiness), and task performance (concentration, ability to play a sport or drive a car). Responses ranged from 0 (least) to 10 (normal) to 20 (most).

Effects of 0.56 g/kg alcohol are compared under double-blind conditions. The appropriate dose of alcohol for each women is calculated according to body weight and mixed with grapefruit juice to yield a ratio of 20% alcohol to total volume (300 ml). The beverage container is a modified thermos bottle with attached straw [49, 57]. Strong alcohol taste and odor cues emitted from the container disguise the plain grapefruit juice in the placebo beverage. All subjects swallow the mixture at a constant rate over 15 min.

After drinking the experimental beverage, women provide breath samples for measurement of blood alcohol levels and complete an alcohol effects scale (SHAS) [77] at 15, 30, 45, 60, 90, 120, 150, and 180 minutes after beverage administration. A hand-held breath monitor estimates blood alcohol levels. Subjects perform psychomotor and cognitive-perceptual tasks at 15, 30, 60, 90, 120, 150, and 180 min. At each assessment time tests are given in random order to limit confounding of alcohol effects with learning effects. The 12 item SHAS [77] measures subjective responses on an unnumbered 36 point scale that ranges from "normal" to "extremely."

For the DSST, subjects responded to a series of digits displayed at random on a computer monitor for 60 seconds by entering a pattern of hyphens and asterisks on an electronic keypad. A computer program recorded the number of trials attempted in 60 seconds and the accuracy of responses. The DSST is one of the most sensitive tests available to measure performance under drug conditions [40, 51].

For body sway assessments, a specially designed instrument with four compression springs placed between two platforms measured subjects' ability to retain a stable upright posture. Subjects focused their eyes on a fixed point (0.6 meters away) while standing with their feet 10 cm apart, and platform oscillations measured spatial disorientation. The number and distribution of sway units for each plane were counted and scores were combined to obtain an overall measure of sway. Body sway also has been found to be affected by alcohol and other psychoactive drugs [47, 76, 85] or observed in youths at risk for alcoholism [37].

FINDINGS

Alcohol expectancies, subjective intoxication levels, and blood alcohol levels

Scores for 3 items were significantly different on 23-item alcohol-effects expectancy scale [77]. FHP women expected to feel less tense, alert, and able to concentrate than FHN women. Mean blood alcohol levels peaked at 80 mg/dl 30 to 60 minutes after drinking alcohol and the rate of disappearance was comparable for FHP and FHN women; blood alcohol levels for each group did not significantly differ, and mean levels were similar. Intoxication ratings ("high" and "drunk") did not significantly differ between FHP and FHN women, although FHP women consistently reported themselves slightly more intoxicated (range: between 5 and 1 rating units). Correlations between blood alcohol levels and intoxication scores were almost perfectly correlated and showed similar patterns for FHP and FHN women.

Performance Measures

The 0.56 g/kg alcohol dose does not affect simple motor tasks such as hand steadiness or rapid finger tapping [cf. 45, 66], but FHP women have shown less impaired Digit Symbol Substitution Test (DSST) and body sway results. Body sway in the lateral plane was marginally significant ($p < .10$) at 30 and 180 minutes after alcohol, anterior-posterior sway differed significantly ($p < .03$) at 60, 90, and 120 minutes after alcohol, and combined sway scores showed significant differences ($p < .05$) at 15, 30, 60, 90, and 120 minutes after alcohol. These results are consistent with differential responses observed in FHP and FHN men [76].

It was established that learning effects for both FHP and FHN subjects had plateaued just prior to alcohol administration. When performance on the DSST was analyzed using change scores, FHP women made significantly fewer incorrect DSST trials 30 minutes after alcohol ($p < .03$), and FHP women had significantly more DSST responses 90 minutes after alcohol ($p < .03$).

DISCUSSION

One possible explanation for apparent differences between FHP and FHN women might be that results simply reflect individual differences in response to alcohol [95] and thus were a chance occurrence. However, our findings are consistent with reports from other laboratories that indicate that some combination of gender-related and family-history related differences operate in alcohol-induced effects. For example, gender differences have been observed in reaction time to a visual stimulus following administration of 0.76 ml/kg (0.6 g/kg) alcohol [87]. Although family history of alcoholism was not controlled, overall young men had faster reaction times at baseline and showed delayed reaction time 15 minutes after alcohol, while young women did not show delayed reaction time until 90 minutes after alcohol, a time at which our DSST results showed impairment.

In a study comparing young men and women, Mills and Bisgrove [61] assessed subjective, cognitive, and motor performance in male light (mean 7.29 g/kg ethanol per month) and female light (mean 7.38 g/kg per month) drinkers. Subjects served as their own controls and were randomly assigned to placebo, low, and high dose alcohol conditions. Subjects were tested at 20, 40, and 60 minutes after receiving an alcohol dose comparable to that used in our study (0.56 g/kg alcohol). Women reported significantly less subjective impairment than men ($p < .05$), and had body sway scores comparable to men (as measured with an apparatus similar to that used by Lipscomb and Nathan [47] and Schuckit [76] but showed more cognitive-perceptual impairment on a divided attention task. Mills and Bisgrove [61] concluded that subjective ratings of alcohol impairment may not be highly correlated with either blood alcohol levels or performance change. However, their study did not control for family history of alcoholism, and menstrual cycle phase was noted at all 3 test sessions but not controlled in data analysis. In another study that included females, Hill and co-workers [39] compared body sway in 9 adolescent girls and boys from "high density" alcoholic families (with alcoholic fathers and two adult male alcoholic siblings) with 10 control children. For ethical reasons, no alcohol was administered. Subjects were given five 1 minute trials with eyes open and eyes closed, and daughters of alcoholics swayed more than sons and controls of both sexes. Use of a single measure and different posture, small sample size, and lack of information about the subjects' age, pubertal status, and possible substance use, limits generalization from this study. However, it is possible to reconcile results by hypothesizing that despite similar patterns of social drinking, greater sensitivity to alcohol effects may lead to greater tolerance t alcohol in young adult FHP women.

In a study of women and men, Sutker and co-workers [84] used a design wherein subjects did not serve as their own controls to compare intoxication levels, negative affect, and physiologic arousal. Alcohol doses were calculated to attain peak levels of 60 mg/dl, and to introduce a "stress" factor, women were tested 26 to 29 days after cessation of menses. For subjects who received alcohol, intoxication ratings did not differ by gender. However, these investigators did not control for family history of alcoholism, and measurements were not obtained at uniform intervals after drinking, but only when subjects attained specific blood alcohol levels. Further, testing during the premenstruum might have confounded premenstrual discomfort with subjective intoxication.

In a study of men, O'Malley and Maisto [68] tested 24 FHP and FHN pairs of men under placebo, low dose (0.82 g/kg) and high dose (1.63 g/kg) alcohol conditions. Groups were assigned to specific doses, and subjects did not serve as their own controls. However, findings were comparable to those reported by Schuckit [77]. FHP men reported less subjective intoxication, irrespective of dose. Results of a multiple regression analysis indicated that the greatest portion of the variance for intoxication levels in FHN men was accounted for by blood alcohol levels, and intoxication levels in FHP men were predicted best by expectancy of alcohol effects [68].

In our study, FHP women expected to feel less tense, less alert and less able to concentrate following 2 alcoholic drinks, while alcohol expectancy effects after 3 alcoholic drinks rated on the same instrument by matched FHP and FHN men [77] showed that FHP men anticipated feeling significantly more tired and depressed, but were less likely to expect heart palpitations. One possible interpretation of differences is that women may generally use different cues to monitor their intoxication level. In comparison to men, young women have been reported to experience more anxiety associated with the social consequences of alcohol consumption [1], and to have different life experiences associated with alcohol [50]. Clearly, effects of female gender and family history of alcoholism on alcohol expectancies require further study.

In our pilot study, we found blood alcohol levels for FHP and FHN women who had consumed 0.56 g/kg alcohol were similar in magnitude to the blood alcohol levels that Schuckit has reported for FHP and FHN men studied after the same alcohol dose [77]. In contrast to FHP and FHN men, however, FHP and FHN women had *lower* SHAS intoxication scores and both groups of women assigned ***similar*** ratings to their subjective assessments of alcohol effects. Thus, men and women may differ in subjective assessment of intoxication [8]. However, it also may be important that each subject received only alcohol or only placebo in our study, in the experiments conducted by O'Malley and Maisto [68], and in the investigations of Sutker and co-workers [84] . In contrast, in the work of Lipscomb and Nathan [47], Mills and Bisgrove [61], and Schuckit [76,77] all subjects received different doses on different days; this type of research design permits comparison of alcohol and placebo effects as experienced by the same subjects. Thus, interactions among gender, menstrual cycle phase, and design factor differences may explain discrepancies among findings reported from studies conducted in various laboratories.

Even though our pilot samples were small, body sway measures after alcohol intake were sufficiently affected by alcohol to distinguish FHP from FHN women in the predicted direction and confirm findings by Schuckit [76] for differences between 34 pairs of FHP and FHN men at +135 minutes after a comparable alcohol dose. Mills and Bisgrove [61] did not find a gender effect in body sway, but reported that women had more cognitive impairment after alcohol than pair-matched men. O'Malley and Maisto [68] did not find significant differences between 24 pairs of FHP and FHN men in number correct on a symbol digit substitution task nor any effects of family history on body sway, but assessments were obtained at different time intervals (15 and 65 minutes after alcohol). In the present study, in the high dose group, and 10 and 35 minutes after drinking placebo or alcohol the low alcohol dose, the number of incorrect DSST trials showed significantly less accurate performance by FHN women 30 minutes after alcohol. i.e. on the ascending limb of the blood alcohol level curve, and significantly fewer trials attempted by FHN women 90 minutes after alcohol.

COMMENTARY

Further tests of effects of family history of alcoholism on sensitivity to alcohol in men and women appear to require careful assessment by a number of physiological, subjective, and behavioral measures. Our results thus far show that a moderate dose of alcohol does not discriminate between the performance of FHP and FHN women on simple motor tasks but is sensitive to measures of body sway and, to a lesser extent, a perceptual-cognitive task (the DSST). Since studies of populations at risk for alcohol problems seek to identify underlying mechanisms that might lead to accelerated development of tolerance and dependence on alcohol, our studies now incorporate divided attention measures [61, 65, 67] to assess systematically other possible cognitive differences between FHP and FHN women.

However, it should be recognized that neither results from studies of male versus female differences in alcohol effects, nor results from studies that contrast of FHP versus FHN subjects are convergent or uniform. In general, the majority of studies show some similarities as well as statistically significant differences in the effects of alcohol on men and women, and results of test batteries given to FHP versus FHN subjects also indicate differential sensitivity to alcohol. These differences may be illuminated or reconciled by continuing studies that compare possible central nervous system changes in responses of prepubertal and adolescent children of alcoholics and controls [89]. Caution in interpretation of those results also is advised, because it is unlikely that all central nervous system changes predict vulnerability to alcoholism [98].

One major concern emerges from this brief review and comparison of selected relevant findings. First, sample sizes that are small may yield results influenced by uncontrolled biases; larger samples generate more stable statistics. However, if findings are to be replicated, then sample selection must be guided by control for interviewing variables. Until there is contrary evidence, it will be necessary to control for menstrual cycle effects in women. A significant trade-off factor, however, is that careful screening and matching inevitably limits sample size and prolongs the data collection process for any research project until a sufficiently large sample can be studied. For this reason it is important to measure several factors in one study. This latter observation underscores another important factor. Results can be compared more readily if equivalent variables and similar testing instruments and apparatus are used by a number of investigators.

Within the last decade children affected by parental alcoholism have come to public attention. Some estimate that about 28,000,000 Americans have at least one alcoholic parent [73]. Special research and policy task forces and commissions have convened to examine the magnitude of problems associated with alcohol abuse and dependence [9]. However, as yet there is no clear consensus on the extent and severity of diminished psychosocial functioning in children of alcoholic parents or the most effective format for treatment [43, 92]. Ethical considerations argue for an interrelationship between prevention and intervention programs.

Further, it must be remembered that numerous factors contribute to inheritance of alcoholism [33, 46, 89], including family environment [4], societal trends favoring increased consumption [17], and alcohol effects on hormone secretion [74]. At this juncture between 40% to 70% of FHP men can be definitively classified by a profile of behavioral and biological variables [74]. No doubt increased understanding will also emerge as more data from different research fields becomes available [cf. 17, 74]. At this time data continue to need interpretation using appropriate models of both psychosocial and biological contributions to the mechanisms of inheritance and transmissions of vulnerability to alcoholism [cf. 17, 74]. Sufficient resources are necessary to continue investigation of factors involved in vulnerability to alcoholism, to evaluate the relative contributions of pertinent risk factors to alcohol problems, and to develop and target appropriate models for education, intervention, and treatment.

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INVITED TESTIMONY TO THE U.S. SENATE COMMITTEE ON GOVERNMENTAL AFFAIRS, MAY 26 1988 -- Robin Room, Scientific Director, Alcohol Research Group, Medical Research Institute of San Francisco, 1816 Scenic Avenue, Berkeley CA 94709.

In a few days, it will have been twenty-five years that I have been working in the field of alcohol studies. It has been a privilege to receive public support to work in a field which is at one and the same time so full of fascination as a research area and so pregnant with possibilities for practical action and change.

The central focus of our research center has been on drinking patterns and problems as they are manifested in the everyday life. To this end, we have conducted a series of surveys of the general population, both in specific communities and for the nation as a whole, studying the correlates and predictors of heavy drinking and of dependence and other drinking-related problems. From this central concern with patterns in the general population, our research has branched out in several directions. We have been deeply involved in longitudinal studies, where individuals in the general population are followed through time to study the patterning of drinking patterns and problems in the lifecourse -- when and how people get into and out of drinking problems. We have studied the community response to alcohol-related problems -- both the informal responses of family and friends and more formal responses such as treatment, counseling or prosecution. We have examined the relations between patterns and worlds of drinking, on the one hand, and societal controls and policies, on the other. And we have extended the frame of our analyses through time in studies of trends and of social change, and across cultures in collaborative studies of the interplay of controls, consumption and consequences in different national histories.

THE SOCIAL LOCATION OF DRINKING AND OF PROBLEMATIC DRINKING

You have asked me to focus on environmental and behavioral risk factors in alcohol problems, and on the interaction of these factors with genetic and other factors. This is indeed a large topic, which I can only tackle here selectively. Let me start by outlining what might be called some "social facts" about heavy drinking and alcohol-related problems -- facts which any explanation, genetic or environmental or developmental, must be able to fit.

Only about two-thirds of adult Americans drink at all, and only one-half drink as often as once a month. These proportions have not greatly changed since the late 1940s. Abstention is particularly common in what we might call the traditionally "dryer" areas of the country -- the southern and prairie states. In the postwar period, the average amount each drinker consumed rose, particularly during the 1960s for the nation as a whole, but also more recently in the traditionally "dryer" areas. The rise in consumption seems to be more attributable to men than to women. In all parts of the country, the rise in per-capita consumption has slowed down or stopped, and since 1981 consumption has been falling slowly for the nation as a whole.

Consumption of alcohol, as for most consumable products, is heavily concentrated; a rough rule of thumb is that 10% of the adult population consumes more than half of the alcohol sold. Richer people and middle-aged people tend to drink more frequently, while younger and poorer people tend to drink more on an occasion, that is, to be more likely to become intoxicated when they drink. In the general population, both heavy drinking and drinking-related problems are more commonly reported by men than by women; we might say that, taken overall, women's biggest problem with drinking is their men's drinking. Problematic drinking is also more common among younger adults than among middle-aged and older people. Among white males, there is a sharp peaking of heavy drinking and drinking problems in the age-group 18-24; for black and Hispanic males, the peak is less sharp and comes later. Those in treatment for alcohol dependence or abuse are on the average much older than this; the average age of treatment clients is usually at least 40. Many people experience some alcohol-related problem without ever receiving treatment; perhaps one-fifth of American adults get into some scrape involving their drinking in the course of their lifetime, while about 3½% report that they have ever been in treatment or gone to Alcoholics Anonymous for their own drinking.

Although there is considerable stability in rates of heavy drinking and of drinking problems in the population as a whole, at the level of individual lives there is a great deal of turnover and change. As the patterns I have just described imply, many young people go through a period of heavy drinking and then "settle down". Some people who experience drinking problems stop drinking altogether. Others have sporadic periods of heavy drinking or drinking problems. Also, while those who end up in treatment for alcohol dependence or abuse have usually accumulated problems with drinking in many areas of their lives, this is not typical for heavier drinkers in the general population. In the general population, having a problem with drinking in one area -- for instance, drunk driving arrests -- is only a modest predictor of having a problem in another area -- for instance, in their family life. While the heaviest drinkers have a greater average number of problems with drinking, those drinking somewhat less account for a greater fraction of the health and social problems related to drinking, since there are so many more drinking at these levels. Even if we could remove from the population all those who fit clinical criteria of alcohol dependence or alcoholism, then, there would still be a very substantial burden on our society of alcohol-related health and social problems.

CHANGES IN DRINKING LEVELS AND PROBLEMS IN A POPULATION

So far, I have mostly described static patterns -- comparisons within the American population when we look at it cross-sectionally. Researchers have often fallen into a habit of viewing drinking and alcoholism only in this way, as if rates of heavy drinking and alcohol-related problems were basically unchanging through time. Some of the most interesting work in recent years has involved opening up the dimension of time and change to systematic study. This has involved work in a number of research traditions: for instance, time-series analyses of health and social statistics, primary-source work in social and cultural history, and analyses of trends in population survey data. Since the U.S. alcohol research effort has particularly emphasized biomedical factors, some of the most interesting work in this social and epidemiological area has been conducted in other countries.

While much of the cross-temporal research has been focused on the understanding of marginal changes, it has also underlined the fact that big changes do happen -- big changes in the level of drinking in a whole population, and in the rates of alcohol-related problems. One of the truly dramatic changes in world history came in the 1830s and 1840s in the United States, with the first wave of the temperance movement. As can be seen in Figure 1, the estimated level of per-capita consumption in the U.S. fell by about half in those years. Then consumption went back up somewhat during the Civil War and at the turn of the century. Many commentators, both at the time and more recently, have focused on this backsliding, concluding that the "moral suasion" of the early temperance movement had been a failure. But the figure shows that in a longer perspective there was a lasting change: U.S. per-capita consumption has never since reached the levels at which people drank when the Republic was young.

Let me show you a couple of other examples of big changes, both in alcohol consumption levels and in rates of alcohol-related problems. Figure 2 shows what happened to cirrhosis mortality in Paris during the two world wars: in these periods of disrupted availability and of privation, in which consumption of alcoholic beverages was much reduced, deaths from liver cirrhosis fell dramatically. On the other hand, Figure 3 and Tables 1 and 2 show what happened to alcohol consumption and to various indicators of alcohol problems in Finland in 1968, when a legislative change made alcoholic beverages much more readily available. As the consumption of alcoholic beverages rose, so did the rates of most alcohol-related problems. Figure 4 and Table

3 show similar data for Poland for 1970-1981, as alcohol consumption rose to a peak in the late 1970s, and then declined both in the heightened morale of the Solidarity period and under the restrictions of the martial law period.

As the Finnish and Polish examples demonstrate, one clear finding is that changes in the levels of alcohol-related problems tend to follow changes in the level of alcohol consumption in a given population. This is true particularly for physical effects of sustained heavy drinking, such as liver cirrhosis and head and neck cancers, but it tends to be true also for other alcohol problem indicators such as rates of casualties and of violent crimes. Although most of these studies are of aggregate patterns for populations as a whole, there are a few studies which directly measure changes in the drinking patterns and problems of individuals in response to such factors as a price increase or an economic downturn. While it is often intuitively assumed that very heavy drinkers would be less responsive to such factors than lighter drinkers, in fact the studies show that heavy drinkers are at least as responsive proportionately (and more responsive in absolute terms) than lighter drinkers.

We can thus conclude that extraordinarily large changes in drinking patterns have occurred in particular populations, sometimes in a relatively short period of time. Holding roughly constant the genetic composition or susceptibility of a population, therefore, the manifestations of heavy drinking and of drinking-related problems can vary very widely. Furthermore, changes in the drinking levels and patterns of the population as a whole seem to affect the behavior of those who are presumably most genetically susceptible -- the heavy and problem drinkers. For drinking behavior and problems, as for human behavior generally, the constraints of our individual genetic composition seem to allow for a very wide behavioral repertoire.

What does this imply for our efforts to reduce the rate of alcohol-related problems in our society? One implication is that we need to understand the conditions under which changes, big and little, occur. To understand the specific genetic mechanisms of susceptibility to or protection from heavy drinking or alcohol problems is an exciting and fruitful scientific task, but the individual testing and counseling this makes possible may be ineffective if it goes against fashionable behavior. I am reminded of an airline's billboard at the end of my street, showing a woman apparently of northern European ancestry sunbathing on a Hawaiian beach, along with the message, "Bake till golden brown". We can tell this woman that the genetically-determined color of her skin means that she has a heightened susceptibility to skin cancer from suntanning. But until our society changes its ideas about the body beautiful, the airline can still count on a positive response to its invocation of a behavior which is for the genetically susceptible a health risk.

PROCESSES OF CHANGE IN AMERICAN DRINKING PRACTICES

What have we learned about how changes in levels and patterns of drinking occur? By studying the "natural experiments" of what happens when there are legislative or cultural changes, we are beginning to draw some conclusions. As for other commodities, alcohol consumers are somewhat responsive to price and other limitations of availability. Some big reductions in alcohol consumption and problems have been the product of extraordinary social crises such as a global war or a major depression -- a finding which I hope is not of much practical help. Other big changes have been driven by religious zeal or despotic controls -- the most recent example would be the drying up of Iran under the rule of the ayatollahs.

In societies like our own, one primary way in which big changes in habitual behaviors happen is through movements seeking social change or spiritual renewal. When de Tocqueville wrote of Americans as a nation of joiners, what he had observed was the first flush of the temperance movement in the 1830s. Today, we find ourselves in another era of growing social ferment over alcohol issues. The most visible signs of this ferment have been political movements which aim to change someone else's drinking -- groups like Mothers Against Drunk Driving and Remove Intoxicated Drivers. But there have also been quieter changes, changes in people's hearts about their own behavior, as many have switched to low-alcohol beverages and then to no-alcohol beverages, as alcohol treatment capacity has grown, and as movements of self-help and mutual-help have grown by leaps and bounds. Alongside the burgeoning fellowship of Alcoholics Anonymous, a whole new manysided movement, of Adult Children of Alcoholics, has emerged and blossomed in the space of less than five years.

What we are witnessing is the unraveling of a half-century-old consensus about alcohol issues. The college kids of the late 1920s and 1930s -- roughly speaking, people of President Reagan's generation -- staged a partly successful generational rebellion against what they called "Victorian morality". Growing up during Prohibition, many students took up drinking, and indeed heavy drinking, as a signal of generational protest and of personal autonomy. Fifteen or twenty years later, with their heavy drinking now becoming a problem in their life, people from the same "wet" generation swelled the ranks of Alcoholics Anonymous as it grew in the 1940s and 1950s. Today, their daughters and sons, now in their 30s or 40s, form the core of the Adult Children of Alcoholics movement.

The college kids of the 1920s and 1930s won their battle against Prohibition. In the wake of Repeal, alcohol problems turned back from being a public issue to being a private anguish. To be thought at all cosmopolitan or forward-thinking, one could not utter a word against drinking. Eventually, in the 1940s, an effort was started to provide for more humane treatment for the growing numbers of alcoholics. Those in this nascent alcoholism movement had to reassure everyone that they had nothing to do with temperance, and were not opposed to social drinking. This was the political and cultural environment in which the idea took root that there were two types of drinkers, social drinkers and alcoholics, marked off from each other at birth or at an early age. Such a view made it possible to provide humane help while not threatening or involving the rest of us "social drinkers".

Meanwhile, the idea that drinking was a part of everyday adult middle-class life continued into the postwar years of growing prosperity. Amid all the attention focused in the late 1960s and 1970s on the drug habits of a new generation of college students, little notice was given to the steep rise in alcohol consumption -- 30% in a decade -- in the same period. Fifteen or twenty years down the road, the heaviest drinkers of this college generation are now providing a new flood of recruits into Alcoholics Anonymous and alcohol treatment programs.

These generational experiences, I would argue, are an important factor in the ongoing shift in the cultural position of drinking in our society. At a more general level, it appears that we are just beyond the crest of one of the "long waves of alcohol consumption", with a periodicity of about 70 years, which researchers have noticed in many industrial societies. The popular movements which we see around us, as well as the pressures for legislative restrictions on drinking, can be viewed as part of a self-corrective process with which a society like the United States comes to grips, however belatedly, with the real social and health costs of levels of alcohol consumption which we used to find tolerable.

SYMBOLISM, DRINKING AND LEGISLATION

Just as legislation played a role in making alcoholic beverages more available and more ubiquitous in American life, so legislation can now play a role in new efforts to prevent alcohol problems. No doubt your hearing on prevention issues will consider this in greater detail. But there is one relevant point I would like to emphasize, which is the strong symbolic aspect of drinking behavior. When we think of motivations for drinking, we tend to think in utilitarian terms -- that people drink to forget their miseries, or as a sedative, or to get high -- or, for that matter, to quench thirst. But drinking, as a sociable behavior carried on in company, is frequently not so much a utilitarian act as it is an expressive and symbolic one. Alcoholic beverages, and drinking behaviors, carry a heavy load of symbolic meanings in our culture. Just to mention the word "champagne" evokes for most of us vivid images. Given the symbolic significance of joining in a toast, or of taking one's turn in "buying rounds", it is a brave drinker who will decline to do so.

Whether one drinks, and how one drinks, is thus often a symbolic statement to an audience. For a nineteenth-century working man, "signing the pledge" to abstain was often a signal of being a serious person concerned about his children's future. For a 1928 coed, drinking in public, along with smoking cigarettes and wearing lipstick, could be a defiant statement of generational identification. For a present-day teenager, having a drink in one's hand can be a signal of daring and a claim on adult status.

For alcohol controls as in other areas, legislation is especially likely to be effective when it backs up and is backed up by popular movements and public sentiment. Thus there is no doubt, for instance, that the raised minimum drinking age laws have resulted in an important decrease in teenage traffic casualties. I would just sound a note of caution about the future. As with Prohibition in the early 1930s, a restrictive law, adopted with substantial social consensus, can offer a tempting target in the long run for symbolic behavior, including flouting the law. Recent accounts of outbreaks of explosive drinking in college fraternities may be an early signal of such a symbolic transformation. We are caught with the dilemma that to outlaw use of a psychoactive drug, even only for part of the population, increases both the power our society attributes to the drug and the symbolic significance of its use. It should be noted that other kinds of preventive legislation -- for instance, an increase in excise taxes or a change in hours of sale -- do not so clearly have this effect.

SOME SUGGESTIONS FOR INITIATIVES

Studies of "natural experiments": Every year, there are a number of significant changes in laws and regulations concerning alcohol in American states and localities. What happens as a result of these changes is rarely systematically studied, and yet the result of such studies are of practical significance, to other jurisdictions as well as to the affected site. Such studies also contribute to our scientific understanding of the antecedents of alcohol abuse and alcoholism. Support of such "legal impact" studies is thus an appropriate and potentially important aspect of the federal government's leading role in research support. At the present level of research funding for NIAAA, support for studies of even a selection of each year's significant changes would be impossible. Facilitating such studies may also require a special funding mechanism, since it is important to gather data in a controlled fashion before a change goes into effect, and changes often occur at short notice.

Collaborative cross-national research: Other national experiences also offer information highly relevant to the U.S. As a nation of immigrants, we are in a particularly strong position to conduct or collaborate on comparative studies of U.S. ethnicities and those in the sending country. A few such studies, for instance among Japanese- and among Mexican-ancestry populations have already been carried out with limited support from NIAAA, and have shed light on processes of acculturation and on the interplay of environmental and cultural factors in drinking. Due to the complexity and the international agreements involved in collaborative cross-national research, special review procedures and funding may be required to ensure a substantial program of U.S. participation in such studies.

Research on alcohol's role in criminal behavior: While NIAAA does currently support a couple of grants in this area, this important area has been under-researched, perhaps because NIAAA's primary mandate is for health-related research. Although a majority of arrests in the U.S. are for alcohol-specific crimes (primarily drinking-driving and public drunkenness), the National Institute of Justice has not supported research on such crimes, and has supported very little research on alcohol in violent crimes.

While the Department of Transportation has in the past supported substantial contract research on drinking-driving and related subjects, only a small amount of basic research in this area is currently under way, primarily supported by NIAAA.

Alcohol issues and federal agencies: As implied by the previous paragraph, there is a serious problem of coordination and of gaps in coverage between federal agencies on alcohol issues. A very wide range of federal agencies have jurisdiction over matters involving alcohol. Each of the many agencies with land-based jurisdiction -- the National Forest Service, the Federal Services Administration, the Army Corps of Engineers, and so on -- are responsible for regulating alcohol sales and consumption and alcohol-related behavior on their land. Financial agencies often affect alcohol policy by their actions: for instance, a high proportion of Small Business Administration minority business loans have been for liquor stores, although minority communities frequently complain about the concentration of such stores in their neigborhoods. Agencies with transport-based jurisdiction have been quite active on alcohol issues, thanks partly to the prodding of the National Traffic Safety Board, but rulemaking by agencies such as the Federal Railroad Administration frequently has a relatively thin research base. The Bureau of Alcohol, Tobacco and Firearms, which has very substantial alcohol jurisdiction, has been historically resistant to accepting any responsibility for alcohol problems prevention in its actions. It has been argued that consumer interests would be better protected if some of BATF's responsibility for alcoholic beverages was transferred to the Federal Trade Commission and was thus subject to regular FTC rules on such issues as ingredient labeling. There is a need for support for research relevant to federal agency actions on alcohol, for mechanisms for the monitoring and coordination of efforts, and for support for staffing to enable NIAAA to play a lead role in such monitoring and coordination.

Alcohol issues and intergovernmental relations: While NIAAA has long maintained relations with state alcohol problems agencies, there seems to be little federal relationship with state alcoholic beverage control agencies. In recent years, state ABCs have taken an increased interest in general alcohol policy and prevention issues; NIAAA has an opportunity to stimulate further interest by moving into an active coordinating and collaborative role. As at the federal level, a very wide variety of state agencies have some involvement in alcohol matters, and an appropriate federal role might be the stimulation of research and demonstration projects relevant to their work. Given the small size of NIAAA's staff, an appropriation would be required to staff such efforts.

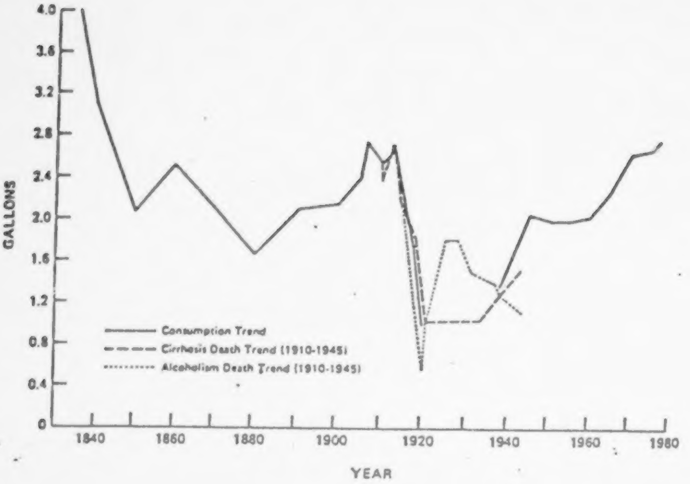


Figure 1 Time trends of U.S. consumption of absolute alcohol, cirrhosis death rate, and alcoholism death rate in the drinking age population, 1830-1977.

Note: Data points are less dense and less reliable prior to 1900. All estimates adjusted to reflect the population 15 years and older.

Sources: Consumption statistics from Hyman et al. (1980); alcoholism death trend from Jellinek (1947-1948); cirrhosis death trend from Bureau of the Census (1975). In estimating Prohibition-era consumption Jellinek favored use of cirrhosis-based estimators; Warburton (1932) used principally alcoholism death rates and production estimates.

From: (Mark H. Moore and Dean R. Gerstein, eds., Alcohol and Public Policy: Beyond the Shadow of Prohibition, Washington, DC: National Academy Press, 1981, p. 35)

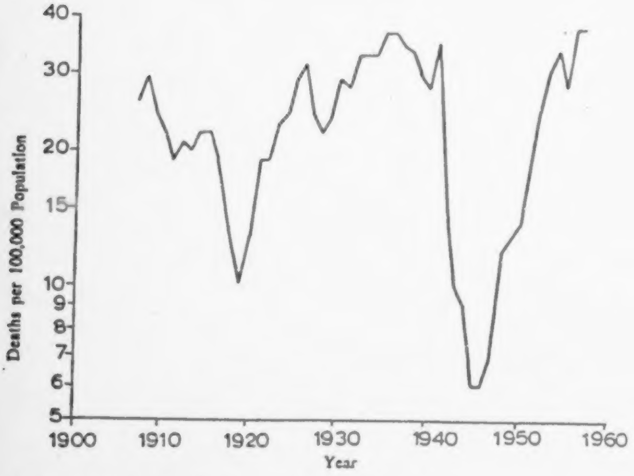
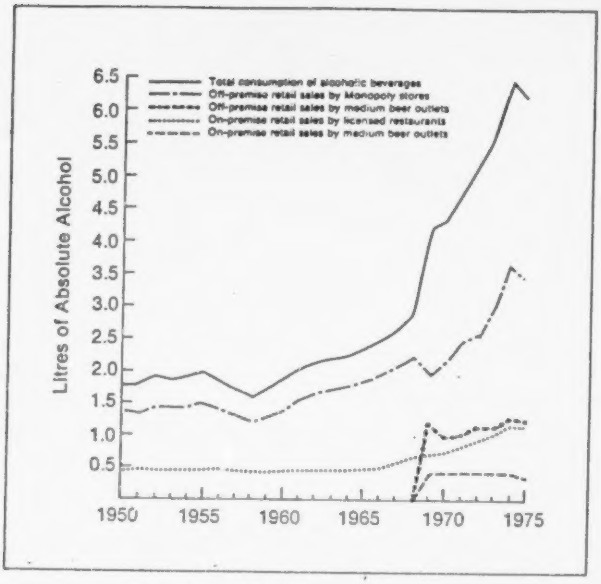


Figure 2 *Liver cirrhosis mortality rates: Paris 1907–56*
Source: Ledermann, 1964.

From: (Kettil Bruun et al., Alcohol Control Policies in Public Health Perspective, Helsinki: Finnish Foundation for Alcohol Studies, 1975, p. 43)

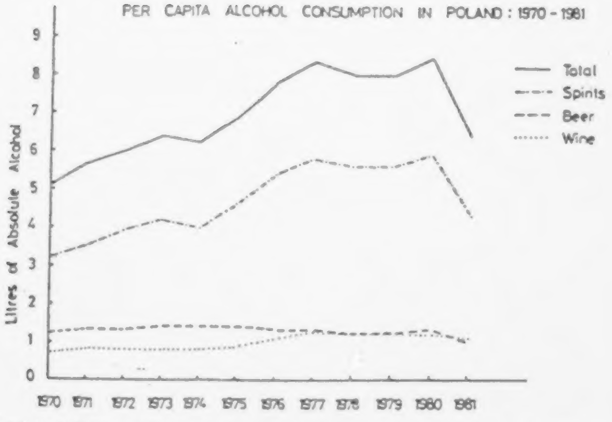
Figure 3 *Consumption of Alcoholic Beverages by Type of Outlet in Litres of 100% Alcohol Per Capita, 1950-1975*



Sources: Alkon vuosikirja 1950-1975; Österberg, 1979a

From: (Klaus Mäkelä, Esa Österberg and Pekka Sulkunen, "Drink in Finland: Increased Alcohol Availability in a Monopoly State", pp. 31-60 in: Eric Single, Patricia Morgan and Jan de Lint, eds., Alcohol, Society and the State: 2. The Social History of Control Policies in Seven Countries, Toronto: Addiction Research Foundation, 1981.)

Figure 4



From: (Ignacy Wald and Jacek Moskalewicz, "Alcohol Policy in a Crisis Situation", British Journal of Addiction 79:331-335, 1981.)

TABLE 1 *Recorded Consequences of Drinking in Finland in 1950, 1960, 1968, and 1975*

	1950	1960	1968	1975
Arrests for drunkenness per 100,000 inhabitants	3,668.0	2,964.0	3,185.0	5,842.0
Crimes of violence per 100,000 inhabitants	153.0	129.0	157.0	283.0
Alcohol-related road traffic accidents per 100,000 inhabitants	20.0	28.5	44.9	75.1
Cases of drunk driving per 100,000 inhabitants	37.5[1]	96.0	147.2	379.0
Deaths from liver cirrhosis per 100,000 inhabitants	2.3[1]	3.3	3.6	6.3
Fatal alcohol poisonings per 100,000 inhabitants[2]	2.2[1]	2.4	5.2	4.3
Deaths from alcoholism per 100,000 inhabitants[2]	0.2[1]	0.2	0.8	2.3
Deaths from alcohol psychosis per 100,000 inhabitants	0.1[1]	0.1	0.1	0.2
Arrests for drunkenness per 1,000 litres of alcohol	16.4	13.1	8.4	8.2
Crimes of violence per 10,000 litres of alcohol	8.9	7.0	5.5	4.6
Alcohol-related road traffic accidents in % in all road traffic accidents	9.4	5.8	7.3	12.9
Cases of drunk driving per 1,000 registered motor vehicles	17.0[1]	11.8	9.3	15.1

[1] 1951

[2] Alcohol poisoning and alcoholism should be dealt with in combination, since according to the office processing statistics these diagnoses are used interchangeably in identical cases of death. More specifically, since 1968 the diagnosis of alcoholism has been used in many cases that earlier would have been classified as fatal alcohol poisonings.

Source: Österberg, 1979b

From: (Klaus Mäkelä, Esa Österberg and Pekka Sulkunen, "Drink in Finland: Increased Alcohol Availability in a Monopoly State", pp. 31-60 in: Eric Single, Patricia Morgan and Jan de Lint, eds., Alcohol, Society and the State: 2. The Social History of Control Policies in Seven Countries, Toronto: Addiction Research Foundation, 1981.)

TABLE 2 *Adverse Consequences of Drinking Reported by Survey Respondents in 1968 and 1976*

Percentage of 15–69-year-old male drinkers reporting who have ever	1968	1976
been arrested for drunkenness	15.4	19.3
had to stay away from work because of alcohol use	10.0	17.8
had to change jobs because of alcohol use	1.1	2.4

Sampling procedures and questions were identical in both surveys. The questions refer to lifetime experience of each consequence of drinking.

Sources: Mäkelä, 1975b; Simpura, 1978a

From: (Klaus Mäkelä, Esa Österberg and Pekka Sulkunen, "Drink in Finland: Increased Alcohol Availability in a Monopoly State", pp. 31-60 in: Eric Single, Patricia Morgan and Jan de Lint, eds., Alcohol, Society and the State: 2. The Social History of Control Policies in Seven Countries, Toronto: Addiction Research Foundation, 1981.)

Table 3 Selected alcohol-related problems 1970–1981

Years	First Admissions to Hospital for Alcoholic Psychoses	First Admissions to Hospital for Alcoholism	Deaths due to Liver Cirrhosis	Sobering-up Station Admissions
1970	1,525	4,253	2,721	214,127
1971	1,659	4,572	2,964	223,926
1972	1,945	4,667	3,102	238,611
1973	2,155	5,144	3,223	247,610
1974	2,069	5,575	3,227	247,069
1975	2,453	5,713	3,462	291,958
1976	2,982	6,441	3,722	307,382
1977	3,635	7,521	3,979	331,227
1978	3,551	7,976	4,195	310,221
1979	3,496	8,139	4,150	302,391
1980	4,389	9,204	4,361	317,768
1981	1,753	6,349	3,831	195,283

Source: Wald, I. *et al.*, 1981. Raport o problemach polityki w zakresie alkoholu [Report on Alcohol Policy] IWZZ Warszawa
Rocznik Statystyczny [Statistical Yearbooks] 1977–1982 GUS Warszawa

From: (Ignacy Wald and Jacek Moskalewicz, "Alcohol Policy in a Crisis Situation", British Journal of Addiction 79:331-335, 1981.)

STATEMENT OF

DR. GEORGE VAILLANT
DARTMOUTH UNIVERSITY
HANOVER, NEW HAMPSHIRE

Senators, members of the press, it's a privilege to be here today. I think I will serve you best if I share with you my unique advantage of having followed the lives of alcoholics from childhood to late middle life.

If I can I want to clarify four paradoxes: 1) that alcoholism is a disease, but one that comes from bad habits, 2) that alcoholism is caused in part by genetic factors rather than psychological factors, but its prevention is largely social and economic. Third, if not treated, alcoholism is an often fatal disease, but hospitalization does not work very well, ~~and~~ Fourth, alcoholism is a very painful disease, but suffering sometimes helps.

If you can look at the first figure you can see that if alcoholism were a simple disease one would expect that alcoholics would drink a large amount and normal people would drink a small amount. Instead, alcohol drinking is smoothly distributed throughout the population, analogous to the way that arteriosclerotic plaques are distributed through the arteries of the people in this room. Once arteriosclerosis reaches a certain point, then you start having problems. Once your daily consumption of alcohol starts to be towards the right-hand side of the curve, you start having problems. But there is no discrete point when alcoholism or arteriosclerotic heart disease begin, and like alcoholism, arteriosclerotic disease comes from bad habits. The Japanese don't have coronary care units; they don't share our habits of being overweight, underexercised, too much cigarette smoking, and eating too many animal fats. ¶ The next Table (II) ~~line~~ makes this point in a slightly different way. On the left-hand side are ~~a different~~ number of alcohol-related problems. The next column shows the number of people in a lifetime study of inner-city men who had different numbers of problems. As you can see, regardless of what definition of alcoholism you use; the individual admitting to have trouble with control (Column 3), whether it meets our standard diagnostic criteria or whether someone in the community actually diagnoses the individual an alcoholic, ~~that~~ it's only a very few people who account for most of the alcohol-related problems. ¶ The next figure (III) shows that, contrary to our usual belief, alcoholism doesn't come from unhappy childhoods. Psychiatric intervention isn't going to help alcoholics very much. If you look at the top part of the figure, you can see that once adult and drinking, individuals who are alcohol-dependent are very different from social drinkers in terms of social class and unemployment and mental health. But in childhood, alcoholics do not differ from controls. ¶ If you look at the next Table (IV) ~~line~~, there's one very important difference in the childhood of alcoholics. They are much more likely to have alcoholic relatives. But the effect of alcoholic relatives seems to be genetic and not environmental. It doesn't matter if you live with them.

~~alcoholic relative~~. Out of our 400 inner-city men, there were 51 men who did not have a multi-problem family, but did have an alcoholic parent, who often didn't live with them. Of these 51 men, 27% became alcoholic. In contrast, of 56 men who had severely socially disrupted childhoods but did not have an alcoholic relative, only 5% of them became alcoholic. So alcoholism is facilitated by genetic factors. TP But if you want to prevent it, it helps if alcohol is relatively expensive compared to discretionary income. If you look at the next figure (V), you can see that in England you could alter whiskey consumption four-fold simply by manipulating its price relative to discretionary income by two-fold. In the last thirty years alcohol has grown progressively cheaper relative to gasoline or the hourly wage, and as alcohol has grown cheaper, partly due to the reluctance of Congress to increase taxation, the consumption of alcohol has steadily increased. TP If you look at the next ~~line~~ Table (VI) you can see another way that social factors are important. Out of our 400 inner-city men, 61% of them had foreign-born parents, but they all shared together the social environment of Boston. Those men who came from Irish or old American stock, those men who if they touched alcohol before they were 21 would break their mothers' hearts, and who, if they weren't dead drunk ten times before they were 22, would break their fathers' hearts, these men were likely to develop alcoholism. In contrast, the men whose parents grew up in Greece and

Italy, who often had been allowed to drink in childhood but whose fathers would have thrown them downstairs if they had come home drunk after age 25--they had very low rates of alcoholism. Thus, ~~by~~ effective legislative intervention ~~is~~ may be to help parents teach their children how to make intelligent drinking decisions, and through counter-advertising do everything we can to make drunkenness, not drinking, socially disapproved. ¶ The next ~~line~~ Table (VII) follows 100 alcoholics for ten years after they had been admitted for detoxification. You can see here that ten years after hospitalization, a third of the alcoholics have died, a third have become sober, and very few are still actively drinking. What this ~~slide~~ table underscores is that alcoholism cuts life expectancy by 15 years; the death rate for alcoholics is three times that of non-alcoholics; and yet if you look at the next ~~slide~~ table, you'll see that hospitalization is not particularly effective in promoting a year of abstinence in alcoholics. This is because alcoholism is analogous to diabetes. Anybody can stop drinking. Remember Mark Twain said he had stopped smoking 20 times. What is important is community treatment to prevent relapse, and we have to help our funding agencies and insurance companies support outpatient relapse prevention treatment, rather than focus our resources on long in-patient hospitalizations. ¶ Finally, in terms of the Supreme Court struggle on whether, because alcoholism is a disease, G.I. benefits should be restored for individuals who lost their benefits through drinking, it is important to make the analogy between alcoholism and leprosy. Lepers lose their fingers, lose their noses, lose their ears, not because the leprosy bacillus eats them away, but because leprosy destroys nerve fibers, and without feeling pain lepers lose their fingers and ears and noses through trauma. Alcoholism interferes with individuals' ability to ~~experience~~ respond to pain. It is important not totally to protect alcoholics from the consequences of their behavior. It is important to help them — within reason — to spoil their own drinking, to hit bottom, as it were. ~~and~~ Anybody who is working on trying to think through the delicate moral issues of the wise care of ~~working with~~ alcoholics would be well advised to attend a couple of Al-Anon meetings to get a sense of how family members and legislators can learn ~~how~~ to "spoil" alcoholics' drinking rather than to "enable" it.

George E. Vaillant MD

George Vaillant's Remarks

Four Points:

1. Alcoholism is a disease, but one that comes from bad habits.

2. Alcoholism is partly caused by genetic factors, but its prevention is largely through social and economic means.

3. Alcoholism, if not cured, is often a fatal disease, but hospitalization plays a very small role in recovery.

4. Alcoholism is a very painful disease, but "defense lawyers," "enabling," and protection from suffering may slow recovery.

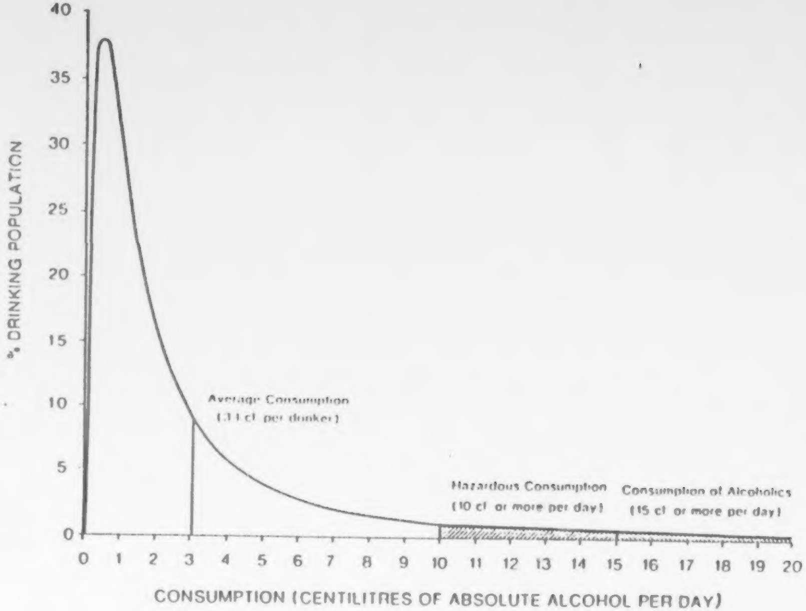


Figure 1.1. Theoretical distribution of adults in Ontario, Canada, according to their consumption of alcoholic beverages (reproduced with the permission of W. Schmidt).

TABLE 1.7. Frequency of alcohol-related problems among the total sample and among those men with commonsense evidence of alcoholism.

Number of problems. (Cahalan and PDS scales combined)	n[a]	*Admits problem with control* (n = 91)	*DSM III alcohol-dependent* (n = 71)	*Diagnosis of alcoholism* (n = 43)
0	157	0%	0%	0%
1–4	83	1	0	0
5–8	48	28	4	0
9–12	38	58	34	11
13–16	28	64	57	25
17–20	21	76	91	67
21–24	12	92	100	75
25–27	9	100	100	100

[a] As in previous tables, the total n is less than 400 because of slightly incomplete data sets.

TABLE 2
Adult Variables That Are More a Result Than a Cause of Alcoholism

Variable	Social drinkers (n = 260)	Alcohol dependent (n = 71)
Adult		
Lowest adult social class (V)	4%	21%
10+ years unemployed	4%	24%
Never completed high school	56%	41%
HSRS < 70	24%	51%
Corresponding childhood		
Lowest parent's social class (V)	32%	30%
Multiproblem family membership	11%	14%
IQ < 90	28%	30%
Childhood emotional problems	32%	30%

Note. HSRS = Health Sickness Rating Scale (Luborsky, 1962).

CHILDHOOD FACTORS IN ADULT ALCOHOLISM

	MULTI-PROBLEM CHILDHOOD	ALCOHOLIC PARENT
51 MEN	0	+ OR ++ (27% ALCOHOLIC)
56 MEN	++	0 (5% ALCOHOLIC)

Figure 2 *Whisky consumption against price per bottle (UK 1950–70)*

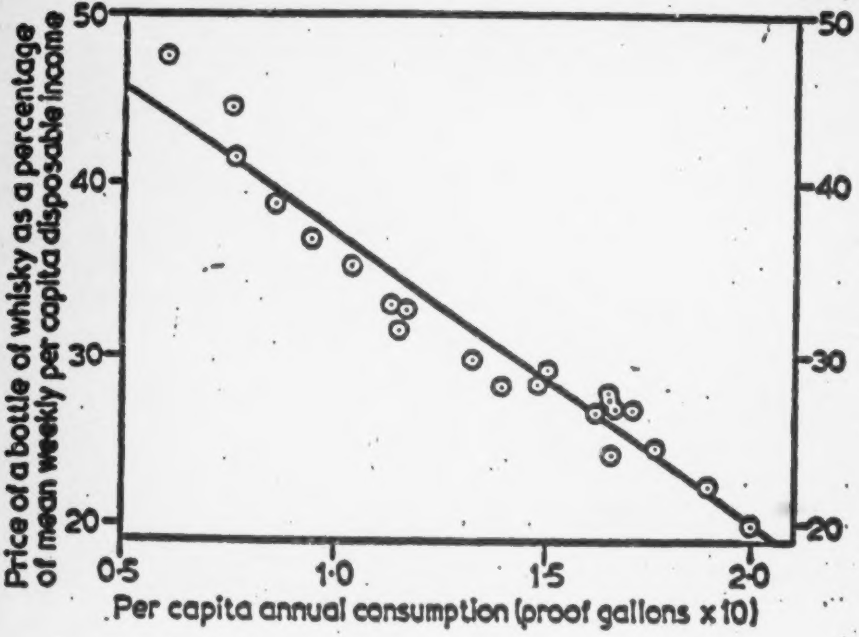


TABLE 2.5. Use and abuse of alcohol .

Ethnic group	Alcohol-dependent (DSM III) (n = 71; 18%)
Irish (n = 76)	28%
Old American (n = 35)	27
Polish, Russian (n = 17)	18
English, Anglo-Canadian (n = 98)	23
Northern Europe other (n = 20)	25
French Canadian (n = 26)	23
Southern Europe other[b] (n = 23)	4
Italian (n = 99)	4
Jewish (n = 6)	0
Chinese (n = 1)	0

[a] Only the 401 men rated on the alcohol differences in this subgroup, men with 2 and 3 on the PDS w

[b] Lebanon, Syria, Turkey, Greece, Portugal

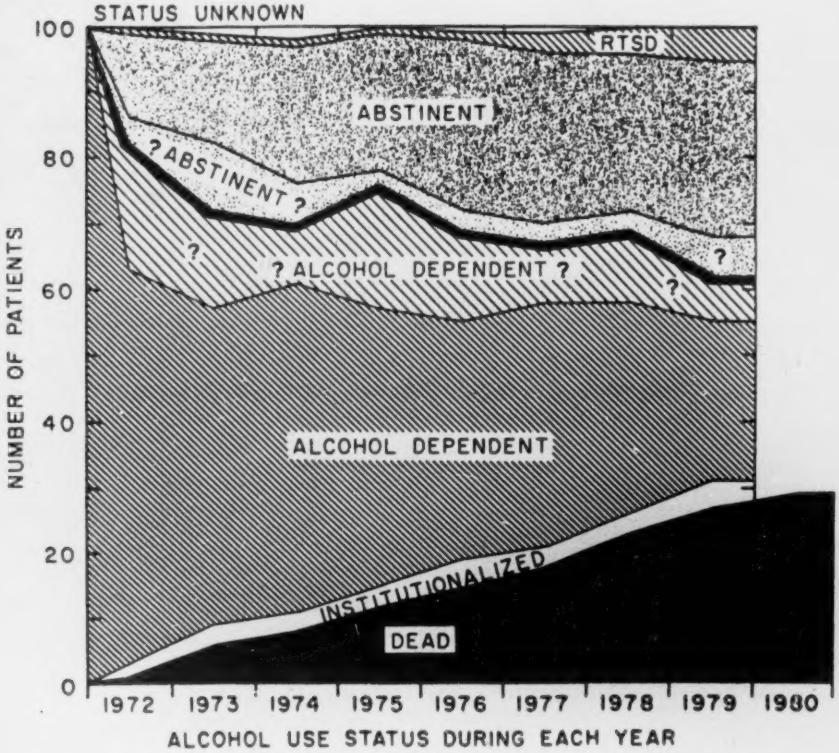


Figure 3.7. Composite posthospital alcohol use histories of the Clinic sample. Abstinent = no alcohol use for more than 51 weeks during a year (corroborated by more than one source). ?Abstinent = evidence for abstinence unreliable, or abstinent for 44–51 weeks. RTSD (return to asymptomatic drinking) = individual drank more frequently than once a month and longer than 12 months

Table 3

Relative Efficacy of Different Modes of Treatment

For 100 Alcoholics	Known "Treatment" Exposures	% followed by 1 yr. of abstinence
Hospital Detoxification	~ 1500	3%
300+ Visits to AA	19	74%

Reference

THE NATURAL HISTORY OF ALCOHOLISM

George E. Vaillant

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The Etiology of Alcoholism

A Prospective Viewpoint

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ABSTRACT: *This article reviews previous prospective studies of alcoholism and then reports multivariate analyses of the data from a new 33-year prospective study of the 456 nondelinquent controls from the Gluecks' delinquency study. The data suggest that presence or absence of South European ethnicity (perhaps as a result of attitudes toward alcohol use and abuse) and the number of alcoholic relatives (perhaps more due to heredity rather than environment) accounted for most of the variance in adult alcoholism explained by childhood variables. Premorbid antisocial behavior also added significantly to the risk of alcoholism. When ethnicity and heredity were controlled, childhood emotional problems and multiproblem family membership explained no additional variance. Thus, the etiological hypotheses that view alcoholism primarily as a symptom of psychological instability may be illusions based on retrospective study.*

In retrospect, it has always seemed clear that alcoholism is a symptom of unhappy childhood and of an unstable or dependent personality. In his classic monograph on alcoholism, Jellinek (1960) states, "In spite of a great diversity of personality structures among alcoholics there appears in a large proportion of them a low tolerance for tension coupled with an inability to cope with psychological stresses" (p. 153). In the most recent edition of the *Comprehensive Textbook of Psychiatry*, Selzer (1980) writes: "Alcoholic populations do display significantly more depression, paranoid thinking trends, aggressive feelings and acts, and significantly lower self-esteem, responsibility and self-control than non-alcoholic populations. Despite occasional disclaimers, alcoholics do not resemble a randomly chosen population" (p. 1629). Other retrospective clinical studies of alcohol abuse have equated the antecedents of alcoholism with those of the oral dependent personality (Blane, 1968).

However, since alcoholism profoundly distorts the individual's personality, his or her social stability, and recollection of relevant childhood variables, retrospective impressions are suspect. In recent years six prospective studies (Jones, 1968, 1971; Kammier, Hoffmann, & Loper, 1973; McCord & McCord, 1960; Robins, Bates, & O'Neal, 1962; Vaillant, 1980) have demonstrated many fallacies in our retrospective conception of the alcoholic's personality. The McCords (1960) observed that, in contradiction to retrospective studies, alcoholics were premorbidly no more likely than controls to manifest phobias, "strong inferiority feelings," more "feminine feelings," "oral tendencies," or "strong encouragement of dependency" from their mothers. Jones (1968) and Loper, Kammier, and Hoffmann (1973) observed that contrary to popular beliefs, alcoholics were, if anything, premorbidly outwardly more self-confident, aggressive, and heterosexual than their peers. Alcoholism also appears to be the cause rather than the result of passive-dependent traits (Vaillant, 1980), of elevations on the D and Pd scales of the Minnesota Multiphasic Personality Inventory (MMPI; Kammier et al., 1973), and of passivity, low self-esteem, and introversion (McCord & McCord, 1960). Could alcoholism also be the cause, not the result, of unhappy childhood, broken families, and personality disorder?

Previous work (McCord & McCord, 1960; Robins et al., 1962) identified premorbid antisocial behavior as an important predictor of alcoholism, but these two important prospective studies were derived from samples that premorbidly exhibited a disproportionate amount of antisocial behavior (Powers & Witmer, 1959; Robins, 1966). To evaluate the previous etiological conclusions, we chose

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to restudy the junior high school boys that the Gluecks (1950) had used as a control group in their monograph, *Unraveling Juvenile Delinquency*. As Table 1 suggests, this second follow-up study offered certain methodological advantages. Originally, the sample of inner-city men was selected in early adolescence for nondelinquency. The men were personally reinterviewed at three points in time: ages 25, 31, and 47—well past the peak age of onset of alcoholism. Besides 19 deaths, attrition was held to 7%, and no subject was completely lost (Vaillant & Milofsky, 1980). The present study identified more alcohol abusers than five of the six previous studies cited in Table 1 combined. In contrast to the previous investigators, the Gluecks had prospectively and painstakingly studied the men's relatives over three generations both for cultural background and for alcoholism—variables that recent reviews (Goodwin, 1979; Heath, 1975) suggest are very important in the etiology of alcohol dependence.

The present report will examine the independent contribution of five major variables to the development of alcoholism. These are ethnic background, alcoholic heredity, antisocial behavior prior to the development of alcoholism, boyhood emotional adjustment, and the presence or absence of familial instability.

The Sample and Methods of Study

The sample included the 456 early adolescent boys studied by the Gluecks between 1940 and 1963 as a control group for their well-known studies of juvenile delinquents (Glueck & Glueck, 1950, 1968). In more recent follow-ups (Vaillant, 1980; Vaillant & Milofsky, 1980; Vaillant & Vaillant, 1981) this group has been called the *core city* sample. This sample had been carefully matched for IQ, ethnicity, and residence in high crime neighborhoods with 456 Boston youths who had been remanded to reform school. Their average IQ was 95 ± 12; eventually 48% graduated from high school or attained high school equivalency certification. Although there were no blacks in the Glueck study, 61% of the boys had one or both of his parents born in a foreign country. Although at age 14 ± 2 years the youths had been chosen for the absence of obvious delinquency, eventually 19% spent time in jail, a datum suggesting that the sample is only modestly biased toward good behavior. Eventually, 7% (a rather high proportion) met Robins' (1966) criteria for sociopathy.

At the time of original study, the boys, their parents, and their teachers were individually interviewed. Public records were searched for evidence of alcoholism, criminal behavior, and mental illness in all first-degree relatives. Multiple social agency reports were available on most families. Over 90% of surviving subjects were reinterviewed at ages 25 and 31 (Glueck & Glueck, 1968). At these interviews alcohol abuse or its absence was specifically recorded. At age 47, a two-hour semistructured interview with a detailed 23-item section on problem drinking was used to reinterview 87% of the surviving subjects. The time frame used was the period from age 21 to age 47. Most of these interviews were performed by individuals with two or more years of experience in alcohol clinics, but unfortunately, significant others were not usually interviewed. In addition to the serial interviews, recent psychiatric, medical, and arrest

TABLE 1
Prospective Studies of the Development of Alcoholism

Study	Age at follow-up	n original sample	n lost to follow-up	n alcohol abusers	n not antisocial abusers	% abusers also dependent[b]	Recent interview	Multiple contacts over time
McCord & McCord (1960)	30–35	325	70	29	11	67	No	No
Robins et al. (1962)	c43	367	81	78[a]	29	34	Yes	No
Jones (1968)	36–38	106	40	6	6	—	Yes	Yes
Jones (1971)	36–38	106	61	3[d]	3	—	Yes	Yes
Kammier et al. (1973)	30–35	—[c]	—[c]	38	38	100	No	No
Vaillant (1980)	55	204	2	26	25	34	Yes	Yes
Core city sample	47	456	56	110	90	67	Yes	Yes

[a] This figure includes men who met Robins' diagnostic criteria for schizophrenia and sociopathy and were therefore excluded from her later (1966) report. Women and controls who abused alcohol are also excluded.

[b] These percentages are our rough estimates of the severity of the alcohol abuse in the reviewed studies based on the authors' criteria for inclusion.

[c] This was a follow-back study.

[d] In an unpublished study by Jones this has been increased to 8.

records were also obtained on most subjects. These records helped to confirm that the subjects were reliable informants and to identify additional alcohol-related problems.

On the basis of a search of social service records and interviews with the boy, his parents, and teacher, clinicians blind to all information after adolescence rated each of the men on the following 12 premorbid variables:

1. *Parental cultural background*: If both parents were foreign born in two different countries, ethnicity was assigned to the place of birth of the father. If only one parent was foreign born, ethnicity was assigned to the birthplace of that parent. If both parents were born in the United States, ethnicity was assigned to the birthplace of the parental grandparents.

2. *Parental social class*: This was the 5-point classification devised by Hollingshead and Redlich (1958).

3. *Parental delinquency*: Criteria similar to the parental alcohol abuse category were employed.

4. *Parental alcohol abuse*: 1 = neither parent alcoholic; 2 = one parent with *minor* evidence (two convictions for alcoholism, mention of alcoholism in official records, or strong suspicion in the case record that was compiled from family interview and multiple social agency reports); 3 = one parent with *major* evidence (two or more criteria for minor evidence and evidence of chronicity) or two parents with minor evidence; 4 = both parents alcoholic, at least one with *major* evidence.

5. *Alcoholic abuse in ancestors*: Alcoholism in first- and second-degree relatives who did not live with subject. If reliable judgment was not possible due to ambiguous or absent familial data, a rating was not made. 1 = no evidence of alcoholism in family; 2 = one relative with minor evidence of alcoholism (defined as in parental alcohol abuse above); 3 = two relatives with minor evidence of alcoholism or one relative with major evidence (defined as in parental alcohol abuse above); 4 = three relatives with minor evidence or two relatives with major evidence.

6. *Boyhood competence*: This 8-point scale (Vaillant & Vaillant, 1981) is intended as a crude scale of ego strength. At entrance into the study, the boys received points for doing regular chores, adjusting well to school socially and academically (controlling for IQ), participating in after-school jobs, and coping with difficulties in their inner city homes. Interrater reliability ranged from .70 to .91.

7. *Childhood environmental strengths*: This is a 20-point scale described in detail elsewhere (Vaillant, 1974) that rated the men by clinical judgment of childhood environmental strengths. This scale gave points for what went well rather than wrong. Interrater reliabilities among three raters ranged from .70 to .89.

8. *Childhood emotional problems*: A subscale of the above scale was used: 2 = good natured, unusually social; 1 = average, no known problems; 0 = emotional problems (e.g., dissocial, feeding problems, very shy, phobias, enuresis after age 8, stuttering, hyperactivity). Interrater reliability was .56.

9. *Childhood environmental weaknesses*: This is a 50-point scale (Vaillant & Vaillant, 1981) based on 25 concrete criteria that reflected the Gluecks' more clinically defined Delinquency Prediction Scale (Glueck & Glueck, 1950). The scale included 5 items reflecting gross lack of family cohesion (e.g., 9 or more social agency contacts, raised for more than 6 months apart from both parents.) Five items reflected lack of affection and 5 lack of supervision by the mother, and 10 items reflected grossly inadequate supervision and affection by the father. If 10 or more of the 25 items were present, the boy was said to belong to a multiproblem family ($n = 62$, or 14%). Interrater reliability was .91.

10. *IQ*: When they entered the study, each subject was given the Wechsler–Bellevue test.

11. *School problems and truancy*: Presence or absence of repeated truancy or disciplinary complaints from teachers, fights with students, and so on.

12. *Infant health problems*: A dichotomous variable made by the Gluecks (1950) regarding whether the mother recollected her son as "cranky, nervous, and fretful as an infant."

On the basis of two-hour interviews at ages 31 and 47 and on the basis of recent mental health and arrest records, clinicians blind to all data gathered before age 31 rated the men at age 47 ± 2 on the following 8 variables:

1. *Health Sickness Rating Scale (HSRS)*: This 100-point scale is described in detail elsewhere (Luborsky, 1962; Luborsky & Bachrach, 1974). Interrater reliability was .89.

2. *Social class*: See Hollingshead and Redlich (1958).

3. *Sociopathy scale*: This scale was based on the presence and absence of the 19 criteria used by Robins (1966) for diagnosing sociopathic personality.

4. *Education*: Since this measure incorporated

all adult education, it was regarded as an outcome measure. Many of the men attended technical school or achieved high school equivalency in their late 20s.

5. *Alcohol abuse in heredity:* This scale was identical to the alcohol abuse in ancestors scale, but ratings were made when the men were aged 47 years, and they included available data on both parental and sibling alcohol abuse. Raters were blind to the men's alcohol use.

Because evidence of alcoholism differs depending on the criteria used for its identification and whether it is viewed from a medical or sociological vantage point, three scales were used for its assessment.

6. *DSM-III scale:* This was a 3-point scale defined by the American Psychiatric Association's (1980) *Diagnostic and Statistical Manual* (3rd ed.; DSM-III) and reflected the medical model for diagnosing alcoholism: 1 = no alcohol abuse; 2 = alcohol abuse; 3 = alcohol dependence (evidence for alcohol abuse and tolerance to or withdrawal symptoms from alcohol). Seventy-one men met the criteria for alcohol dependence.

7. *Cahalan scale:* This scale was the 11-item scale devised by Cahalan (1970) and co-workers to assess the national prevalence of alcohol abuse; it reflected a sociological model.

8. *Problem Drinking Scale (PDS):* The Problem Drinking Scale (Vaillant, 1980) was an equally weighted 16-point scale devised to combine the emphasis of the DSM-III on physiological dependence and the emphasis of the Cahalan (1970) scale on social deviance. Of the 456 men, 442 could be rated with some confidence and 400 with great confidence on this scale. Focusing on the 400 best studied cases, men with 0 to 1 symptoms on the scale were, by definition, *social drinkers* (*n* = 256). Men with 4 or more problems were defined as *alcohol abusers* (*n* = 110).

Premorbid Predictors of Alcoholism

Our sample was interviewed at four points over a 33-year time span. We were able at least to locate all of our surviving subjects after age 40, and we could make a reasonable judgment regarding the presence or absence of alcohol abuse in 442 of the men (Vaillant & Milofsky, in press). We obtained essentially complete data sets on 400 surviving men and personal interviews on 367 of our surviving subjects. (Nineteen men died before 40 and were excluded from most data analyses.) Available evidence (Vaillant, 1982) suggests that attrition was more common among the antisocial and emotionally unstable than among the alcohol abusers.

TABLE 2
Adult Variables That Are More a Result Than a Cause of Alcoholism

Variable	Social drinkers (n = 260)	Alcohol dependent (n = 71)
Adult		
Lowest adult social-class (V)	4%	21%
10+ years unemployed	4%	24%
Never completed high school	56%	41%
HSRS < 70	24%	51%
Corresponding childhood		
Lowest parent's social class (V)	32%	30%
Multiproblem family membership	11%	14%
IQ < 90	28%	30%
Childhood emotional problems	32%	30%

Note. HSRS = Health Sickness Rating Scale (Luborsky, 1962).

Table 2 illustrates that in our sample the differences frequently observed between alcoholic and nonalcoholic subjects to which retrospective studies often attribute etiological significance—differences in social class, unemployment, eventual educational achievement, and mental illness—in fact appear *after* the development of alcoholism. Although as adults our alcohol-dependent subjects appeared to have personality disorders and to be socially inadequate, as children they were no less underprivileged than their peers who were to drink socially as adults; they were no less intelligent; and they manifested no more evidence of childhood emotional vulnerability. Other childhood variables that did not correlate with subsequent alcoholism included the quality of maternal affection (a subscale of the childhood weakness scale) and the number of mentally ill relatives.

Table 3 puts into perspective the relationship between childhood variables that did correlate with alcoholism and those that correlated with other facets of adult outcome. The purpose of the table is to tease out differences in the etiology of alcoholism, the etiology of sociopathy (as defined by Robins, 1966), and the etiology of poor adult mental health (as defined by the Health Sickness Rating Scale; Luborsky, 1962). The first four childhood variables—childhood environmental strengths (Vaillant, 1974), boyhood competence (Vaillant & Vaillant, 1981), childhood emotional problems, and IQ—significantly predicted independent ratings of adult mental health made 33 years later. These four variables predicted sociopathy less well, and they were only marginally correlated with adult alcoholism. The next four variables—delinquent parents, multiproblem families (childhood environmental weaknesses), poor infant health, and premorbid antisocial personality—correlated most powerfully with sociopathy, less so with alcoholism, and only marginally with subsequent mental health. Finally, the last four variables seemed significantly associated with adult alcoholism but not with sociopathy or poor mental health. (As can be seen in Table 3, the association between alcoholism and premorbid etiological variables was not really affected by whether alcoholism was defined according to the medical model of the DSM-III or the social deviance model of Cahalan.)

The next three tables examine some of the variables in Table 3 in greater detail. As Table 4 illustrates, the 71 men with several alcoholic relatives were three times more likely than the 178 men with no alcoholic relatives to develop alcohol dependence. This relationship remained significant but not as dramatic if parents and siblings (who could exert environmental effects, genetic effects, or both) were excluded from the comparison and the more conservative alcohol abuse in ancestors scale was substituted.

Alcoholism, especially in young men, may be a cause as well as a symptom of antisocial behavior. Table 5 presents the association between alcohol dependence and premorbid truancy and school behavior problems at age 14 ± 2 (such misbehavior antedated alcohol abuse in all but two of the men). Men with serious premorbid behavior problems were four times as likely to develop alcohol dependence, but since there were only 16 men with severe behavior problems, premorbid antisocial behavior cannot be invoked as playing a major etiological role for most of the 71 alcohol-dependent men in the study.

Although the men themselves were native born and shared a common social environment, the cultural mores of their parents (half of whom were foreign born) differed greatly. Such differences in parental ethnicity were highly correlated with the men's subsequent use of alcohol. Table 6 contrasts the culture in which the men's parents were raised with the likelihood of the development of alcohol

TABLE 3

Childhood Variables Predicting Poor Adult Mental Health, Sociopathy, and Alcoholism

Childhood variable	DSM-III (n = 395)	Cahalan scale[a] (n = 399)	Problem drinking scale[b] (n = 442)	HSRS (n = 378)	Sociopathy[c] (n = 430)
Mental health predictors					
Childhood environmental strengths	− 18***	− 15**	− 14**	21***	− 17***
Boyhood competence	− 12**	− 14**	− 11**	24***	− 22***
Childhood emotional problems	*ns*	*ns*	*ns*	− 19***	.13**
IQ	*ns*	*ns*	*ns*	15**	− 10*
Sociopathy predictors					
Childhood environmental weaknesses	15**	16***	14**	− 10*	18***
Delinquent parents	*ns*	*ns*	*ns*	− 10*	13**
Truancy/school behavior problems	20***	20***	19***	*ns*	.35***
Poor infant health	11**	10*	09*	12**	.17***
Alcoholism predictors					
Alcoholism in parents (1945)	20***	17***	20***	*ns*	10*
Alcoholism in ancestors[d]	14**	15**	10*	*ns*	*ns*
Alcoholism in heredity (1978)	26***	28***	23***	*ns*	11**
Cultural background[e]	− 27***	− 25***	− 27***	*ns*	− 12**

Note. Pearson product-moment correlation coefficients were the statistics used. HSRS = Health Sickness Rating Scale (Luborsky, 1962); DSM-III = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., American Psychiatric Association, 1980).

[a] See Cahalan (1970).

[b] See Vaillant (1980).

[c] See Robins (1966).

[d] Excluding parents.

[e] For purposes of statistical depiction, Irish ethnicity was assigned a weight of 1, old American, Canadian, or British ethnicity, a weight of 2, other Northern European ethnicity, a weight of 3, and Southern European ethnicity, a weight of 4 (see Jellinek, 1960). For purposes of multiple regression and statistical analysis, a given ethnicity is treated as a dummy variable.

* $p < .05$. ** $p < .01$. *** $p < .001$.

dependence among the men themselves. The 75 men of Irish extraction were seven times more likely to manifest alcohol dependence than the 130 men of Italian, Syrian, Jewish, Greek, or Portugese extraction.

Admittedly, the relationship between alcohol use and culture is extremely complex (Greely & McReady, 1980; Heath, 1975; Marlatt & Rohsenow, 1980; Stivers, 1976), but in this study the relationship is reduced to a single common denominator—cultural characteristics of alcohol use that empirically affect the likelihood of alcohol dependence. Thus, ethnicity was crudely scaled to contrast cultures that forbid drinking in children but condone drunkenness in adults with those cultures that teach children how to drink responsibly but that forbid adult drunkenness (Jellinek, 1960; Pittman & Snyder, 1962). Space does not permit full elaboration of the rationale for this division. The non-Moslem Mediterranean countries have no sanctions against children learning to drink but have strong sanctions against drunkenness in adults. On the basis of Snyder's (Pittman & Snyder, 1962) work, the eight Jews in the study were included with the Mediterraneans, and the one Chinese was excluded. Conversely, the Irish and North American cultures forbid drinking in adolescence, have flirted with total prohibition for a century, but give tacit or explicit approval to drunkenness in male adults. For purposes of statistical depiction, other Northern European countries were assigned an intermediate position. Jellinek (1960) points out in some detail that although France teaches children responsible drinking, it also condones drunkenness and alcohol use independent of meals.

The interaction between parental culture and familial alcoholism was interesting. Among men of Irish extraction the presence of alcoholic relatives only slightly increased the risk of alcohol dependence in the subjects. Many Irish subjects with alcoholic relatives became lifelong teetotalers. Among other ethnic groups (both among the Southern Europeans in whom alcohol abuse was rare and among the Northern Europeans and old Americans in whom it was common), alcohol de-

TABLE 4

Number of Alcohol Abusing Relatives and Development of Alcohol Dependence Among Core City Men

Scale	DSM-III alcohol classification: No abuse (n = 267)	Abuse without dependence (n = 60)[a]	Dependence (n = 71)
Alcohol abuse in heredity			
No relatives (n = 178)	78	12	10
1–2 relatives (n = 149)	60	20	20
Several relatives (n = 71)	52	14	34[c]
Alcohol abuse in ancestors[b]			
No relatives (n = 213)	73	31	14
1–2 relatives (n = 133)	63	18	19
Several relatives (n = 52)	58	13	29[c]
Alcohol abuse in parents			
Mother alcoholic (n = 36)	50	14	36[c]
Father alcoholic (n = 149)	58	14	28[c]
Neither (n = 244)	75	15	11
Total sample (n = 398)	67	15	18

Note. Rows, not columns, total 100%. DSM-III = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; American Psychiatric Association, 1980). Values are in percentages.

[a] Alcohol abuse as defined by the DSM-III criteria was a somewhat more inclusive category than alcohol abuse defined by having 4 or more symptoms on the Problem Drinking Scale (Vaillant, 1980).

[b] Excluding parents and siblings.

[c] $p < .01$ by chi-square test.

TABLE 5

Relationship of Premorbid Antisocial Behavior to Development of Alcoholism

DSM-III classification	Truancy or school behavior problems: Absent (n = 381)	Present (n = 16)
No alcohol abuse	69	31
Alcohol abuse	15	13
Alcohol dependent	16	56

Note. DSM-III = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; American Psychiatric Association, 1980). All $ps < .001$ by chi-square test. Values are in percentages.

TABLE 6

Relationship Between Parents' Culture and Development of Alcohol Dependence

DSM-III classification	Culture of parents: Irish (n = 75)	Other[a] (n = 193)	Mediterranean (n = 130)
No alcohol abuse	59	58	86
Alcohol abuse without dependence	13	19	10
Alcohol dependent	28	23	4

Note. Values are in percentages. DSM-III = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; American Psychiatric Association, 1980). All $ps < .001$ by chi-square test.

[a] Canadian, American, Northern European.

TABLE 7
Contribution of Selected Premorbid Variables to Number of Alcohol-Related Problems and Sociopathic Behaviors

	Alcohol-related problems		Sociopathic behaviors[a]	
Variable	% explained variance	beta weight	% explained variance	beta weight
Alcoholism in heredity	7.6	.19	1.3	.05
No Mediterranean ethnicity[b]	3.5	.20	1.0	.11
School behavior problems[b]	3.0	.18	8.0	.30
Boyhood competence	1.3	.07	2.3	.14
Childhood environmental weaknesses	.1	.05	2.1	.07
Poor infant health	.1	.10	2.9	.17

[a] See Robins (1966).
[b] To minimize the variance that they could explain, these two variables were entered into the multiple regression after the other four variables.

pendence occurred five times as often in men with several alcoholic relatives as it did in men with no alcoholic relatives.

Finally, we do not wish to imply that psychological stress and vulnerability are irrelevant in alcoholism, but only that a prospective design and a multifactorial design greatly diminish their importance.

The facts are by no means all in, but primary prevention may be served if our schools, churches, and legislatures pay less attention to prohibition and more attention than they have in the past to relevant cultural differences in attitudes toward alcohol use and abuse. Primary prevention may also be served if individuals with many alcoholic relatives learn that their enemy is neither alcohol per se nor their genetic moral fiber, only that their constitution allows them to "hold" their liquor rather better than less genetically vulnerable individuals (Goodwin, 1979).

Analysis of the Findings

The correlations in Table 3 reflect the essential findings of the study: Except for the Childhood Environmental Strengths scale, the premorbid variables that most clearly predict alcoholism do not predict mental health and vice versa. Although correlations in Table 3 seem low, it must be remembered that 33 years separate the childhood and adult ratings. In an exhaustive review of the prospective literature on childhood-to-adult studies, Kohlberg, LaCrosse, and Ricks (1972) report that if IQ is excluded, correlations of .2 to .4 are the highest observed between ratings of childhood and adult behavior. Wordsworth and Freud notwithstanding, prospective study of life spans has taught us that "the child is father of the man" in a most limited fashion.

To be fully understood, further analysis of the Table 3 variables is necessary. For example, can the increased risk of alcoholism in children from multiproblem families be explained by the fact that many of the problems in such families are the result of alcoholic parents and grandparents? For the purposes of multiple regression analysis, it seemed appropriate to combine the Cahalan scale and the PDS to produce a single scale ranging from 0 to a maximum of 27 alcohol-related problems. Table 7 examines the independent contribution to the total number of alcohol-related problems for each of the potential etiological variables identified in Table 3. Alcoholic heredity, school behavior problems, and not being brought up in a Mediterranean culture (entered into the multiple regression analysis as a dummy variable) each made an important contribution to the explained variance in subsequent alcohol problems. In addition to these three variables, boyhood competence made a small additional independent contribution to alcohol abuse. If the number of alcoholic relatives is controlled for, multiproblem families are not associated with an increased number of alcohol-related problems, but they are associated with an increased number of antisocial problems. Similarly, in Table 3, childhood environmental strengths was significantly correlated with both alcoholism and sociopathy, but entering the variable in the regression equation in Table 7 explained less than 1% additional variance.

The present data suggest that having many alcoholic relatives is associated with an increased risk of alcoholism, but the data cannot distinguish the effect of genes (having a blood relative with alcoholism) from that of environment (being raised by an alcoholic parent or surrogate parent). However, review of all available cross-fostering studies that have distinguished heredity from environment (Bohman, 1978; Goodwin, 1979; Goodwin et al., 1974; Schuckit, Goodwin, & Winokur, 1972) suggests that alcoholic biological relatives (even if physically absent) apparently contribute far more to the observed increased risk for alcoholism in children than alcoholics in the environment. These observations would help explain why the association of disorganized families with subsequent alcoholism disappears if familial alcoholism is controlled.

Another variable that contributed significant independent variance to the likelihood of adult alcohol abuse was premorbid antisocial behavior. Certainly, antisocial inner city youths are at greater risk for developing all types of drug abuse (Chein, 1964; McCord & McCord, 1960; Robins, 1966, 1974), and the rate of alcohol abuse that the Gluecks observed among their 31-year-old delinquents was higher than the rate observed among the controls who make up this study (Glueck & Glueck, 1968). Jones (1968) and unpublished findings from a longitudinal study of college men (Vaillant, 1980) agree that middle-class adolescents who become alcoholics also exhibit rapid tempo and greater impulsivity than their peers.

However, the relationship between alcoholism and antisocial personality is a two-way street. Antisocial symptoms are often a result rather than a cause of alcohol abuse. Thus, if only 9 of the 110 core city alcohol abusers were antisocial prior to alcohol abuse, at least 12 of the 32 adults who eventually met five or more of Robins' criteria for sociopathy were alcohol-dependent before meeting the criteria for sociopathy. In the same vein, the differences in the etiology of sociopathy and alcohol abuse that were suggested in Table 3 were further supported by multiple regression analysis. The three variables in Table 7 that account for the greatest independent variance in adult sociopathy are school behavior problems, boyhood competence, and poor infant health. The ethnic and familial factors that were important in alcohol abuse were unimportant in sociopathy.

The possibility that the childhood syndrome of "minimal brain damage," or so-called hyperactivity, could be an etiological link shared by both adult alcoholism and sociopathy is intriguing (Cantwell, 1972; Tarter, McBride, Buonpane, & Schneider, 1977), but in presenting their evidence for the possibility, Goodwin, Schulsinger, Hermansen, Guze, and Winokur (1975) admit that their hypothesis is still "based on retrospective information and small numbers" (p. 349). Unfortunately, when they were first studied, the core city sample were not only already past the age of maximum risk for hyperactivity, but the syndrome was not to be delineated for another 20 years.

As an independent check on the conclusions of Table 7, the contributions of the six variables to alcohol abuse as defined by the DSM-III, by the PDS, and by the Cahalan scale were separately examined. The multiple regression results did not differ appreciably from those in Table 7 either in percentage of explained variance or in beta weights.

As a second independent check, all the 113 psychosocial variables originally coded dichotomously by the Gluecks (1950) were assessed by their value in predicting subsequent alcohol abuse. Six Glueck items—father's alcoholism, marital conflict, lax maternal supervision, many moves, no attachment to father, and no family cohesiveness—were very significantly ($p < .001$) correlated with the subsequent development of alcohol abuse in study subjects. These six dichotomously rated Glueck items—almost identical to the predictor variables identified by the McCords (1960) as having the greatest etiological impact in alcoholism—were used to create a 7-point composite predictor variable. If this composite variable were entered first into the multiple regression analysis shown in Table 7, it could explain 7% of the observed variance in subsequent alcohol-related problems. However, if this were done, the number of alcoholic relatives and the dummy variable, Mediterranean culture background, could still explain an additional 8% of variance. Conversely, if alcoholic heredity, truancy, and ethnicity were entered first into the regression equation, then the six Glueck variables explained only 1% of further independent variance.

As a check to our conclusions that Syme (1957) and Cahalan and Room (1972) are correct in asserting that specific personality facets are not major etiological factors in alcoholism, we compared remitted core city alcoholics with nonalcoholics (Vaillant, 1982). Former alcoholics with current stable abstinence of more than three years duration did not significantly differ from lifelong social drinkers in terms of current mental health, nor did they differ from the unremitted alcoholics in terms of premorbid risk factors.

Conclusions

Obviously, a life-span perspective is only one of a myriad of techniques required to elucidate a subject as complex as alcohol abuse. To achieve the advantages of a three-decade prospective study, methodological compromises were necessary that may render our conclusions inapplicable to other populations. First, although very similar findings to those in this article were derived from a parallel 40-year prospective study of 204 highly educated upper-middle-class white males (Vaillant, 1980), the present inner city, poorly educated sample included no women, blacks, Native Americans, or rural males. Second, by excluding the most severe delinquents, the Glueck sample minimizes the contribution of premorbid social deviance to alcoholism. However, it is not news that sociopaths have high rates of psychoactive substance abuse. Studies to complement the follow-ups of disproportionately antisocial youth by Robins and the McCords are needed. Third, the enormous number of data available on each subject and the long duration of the study necessitated both composite and simplified global assessment and dependence on redundant, naturalistic (rather than specific) experimentally controlled observations. Examination of discrete items possible in a monograph (Vaillant, 1982) or in a short-term study is not possible in this article. Thus, ethnicity, family history of alcoholism, and childhood weaknesses reflected simplified approximations based on available data. If such methods fail to do full justice to the complexity of the variables in question, nevertheless like aerial photography, they add fresh perspective to our understanding of the alcoholic's landscape.

We do not wish to imply that in adult life other variables do not possess etiological importance. Demographic variables (Cahalan, 1970), occupation (Plant, 1979), social peer groups (Jessor & Jessor, 1975), legal availability (Terris, 1967), societal instability (Pittman & Snyder, 1962), affective illness (Winokur, Clayton, & Reich, 1969), attribution and expectancy effects (Marlatt & Rohsenow, 1980), and other, as yet unidentified, factors certainly contribute independent variance to the development of alcohol dependence.

Nevertheless, if we wish to understand alcoholism, a prospective vantage point is essential. Available prospective studies suggest that if one controls for antisocial childhood, cultural attitudes toward alcohol use and abuse, for alcoholic heredity, and most especially for the *effects* of alcohol abuse then many of the childhood and adult personality variables to which adult alcoholism has traditionally been attributed will appear as carts and not horses.

Indeed, our findings suggest that even when they employed prospective design, previous investigators may have sometimes erroneously interpreted their data to support the retrospective illusion that alcoholism *must* be a symptom of personality disorder (Jones, 1971; Lisansky-Gomberg, 1968; McCord & McCord, 1960; Robins, 1966). For example, in concluding that dominant father, "immigrant" Catholicism, and low social class protected against the development of alcoholism, the McCords failed to note that in Cambridge and Somerville in 1940, such individuals were predominantly first- and second-generation Italians. Italian-American drinking practices, rather than other characteristics, may have protected their children from later alcohol dependence. Similarly, although the 51 alcoholic fathers and the 15 alcoholic mothers in the McCords' study parented a disproportionate number of alcoholic subjects, the McCords wrote that "evidence for an hereditary explanation of the disorder was unlikely" (p. 28). Until more definitive cross-fostering studies (Goodwin, 1979) were available, few investigators could believe that the association between parental and child alcoholism might be more hereditary than environmental. When we are on land and view fish under water, we tend to trust our eyes and not the student of parallax who tells us that the location of the fish is an illusion. Similarly, the parallax of retrospective vision distorts what our "eyes" tell us about the etiology of alcoholism.

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POTENTIAL BIOLOGICAL MARKERS IN INDIVIDUALS AT HIGH RISK FOR DEVELOPING ALCOHOLISM

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POTENTIAL BIOLOGICAL MARKERS IN INDIVIDUALS AT HIGH RISK FOR DEVELOPING ALCOHOLISM

INTRODUCTION:

In the past two decades a plethora of scientific evidence has been accumulated to support the notion that alcoholism is a familial disorder with the presence of genetic factors. It is now well established that sons of alcoholic fathers are approximately four times more likely to develop alcoholism compared to the male offspring of nonalcoholics (Goodwin, 1979). Indeed, the evidence supporting the contribution of genetic factors in alcoholism is sufficiently compelling to warrant a search for markers of the genetic influence.

It should be noted that the antecedent factors or predisposition toward alcoholism might well be assessed with the use of various strategies. It may be possible to study genetic factors by comparing abstinent alcoholics to control subjects or by comparing abstinent alcoholics with a positive family history to abstinent alcoholics with a negative family history of alcoholism. While this particular strategy may yield interesting findings the interpretation of the data may be confounded by significant differences in drinking history, differential age of onset of alcohol abuse as well as numerous other clinical variables such as number and severity of withdrawal episodes, alcohol-related medical complications, etc.

A strategy typically used in the study of genetically determined disorders is the use of large family pedigrees. This powerful approach enables the investigator to study large families with alcoholism and compare them to control families where alcoholism is absent. This particular strategy is most compelling if one studies genetically determined diseases such as Huntington's disease where the phenotypic expression is easily characterized and specific phenotypic markers are readily available. This approach is indeed most powerful to examine the pattern of inheritance. However this method is most difficult to implement without the availability of valid and reliable biological or behavioral markers.

In the last few years investigators concerned with the potential role of genetic factors in alcoholism have used the high risk paradigm. Comparing the sons of alcoholic fathers and nonalcoholic fathers is a useful approach for examining potential biological or behavioral factors associated with a predisposition for alcoholism. This particular strategy has been utilized in biological as well as behavioral investigations, and has already yielded promising data from various studies.

EEG:

Some of the most promising investigations of potential biological markers for a predisposition to alcoholism come from various laboratories which study the electrical activity of the brain. In the last two decades a number of investigators have observed that the resting state electroencephalographic (EEG) activity recorded from awake male alcoholics manifests excessive high frequency activity (Beta, fast EEG) and appears to be somewhat deficient in the appropriate production of lower frequency EEG activity such as alpha (for review see Begleiter and Platz, 1972). Moreover, the production of fast EEG activity has been demonstrated to be genetically transmitted (Vogel, 1970; Young, Lader and Fenton, 1972; Propping, 1977).

These interesting EEG findings and the population genetics data in alcoholism suggested that an excess of fast EEG activity should be present in sons (but not in daughters) of alcoholics. Gabrielli, Mednick, Volavka, Pollock, Schulsinger and Itil (1982) tested this hypothesis in a sample of 265 Danish children. Among this large group of children the investigators found 27 children of alcoholics and compared them with children of normal parents. As expected, they observed that male children of alcoholics manifested excessive fast EEG activity compared to male controls. In addition they report that female offspring of alcoholics do not show this increase in fast EEG activity.

As early as 1968, Naitoh and Docter reported that the administration of alcohol to alcoholic subjects had primarily a stimulant-euphoriant effect, with the subjects reporting that they felt more alert and sociable than controls. Naitoh and Doctor (1968) suggested the intriguing hypothesis that alcoholics drink in order to achieve the psychological state which is associated with an increase and slowing of alpha activity. A number of fundamental questions were raised by the observations of Naitoh and Doctor (1968), as follows:

(1) Are individuals with poor alpha activity at greater risk to become alcoholics?

(2) Are individuals at high risk for alcoholism more likely to show increased production and slowing of alpha after alcohol administration?

Answers to these questions might help delineate the possible relationship between biological markers and subsequent alcohol abuse or alcoholism.

A study conducted by Pollock, Volavka, Goodwin, Mednick, Gabrielli, Knop and Schulsinger (1983) attempted to study the relationship between alcohol intake and alpha frequency and production in high risk individuals. Pollock and co-workers studied alpha activity in a group of males 19 to 21 years of age. They studied a group of 44 sons of alcoholics and a group of 28 controls matched for age and social class. They recorded the EEG 30, 60 and 120 minutes after the administrations of 0.5 g/kg of 95% ethanol. Blood alcohol level was taken at 40 and 130 minutes after alcohol administration. It is important to note that blood alcohol levels did not statistically discriminate between high risk and low risk individuals. However the quantitative analysis of alpha activity revealed significant differences between the two groups. The high risk group demonstrated greater increases of slow alpha activity and greater decreases of fast alpha activity after alcohol ingestion than the control subjects. The high risk subjects also demonstrated greater decrease in alpha frequency after alcohol administration compared to control subjects.

The question of whether predrinking characteristics of the EEG pattern (or the initial brain state) partially determines whether alcohol will have a stimulating or depressant effect would appear to be a fruitful area of investigation particularly within the high risk paradigm. The potential relationship between a specific subjective state and a particular characteristic of brain activity which modulates either the craving for, or the response to alcohol may have fundamental implications for both the search for biological markers as well as the search for drugs capable of inducing the appropriate changes in the brain state responsible for abusive alcohol intake.

EVOKED POTENTIALS:

While the EEG is a measure of the spontaneous electrical activity of the brain, evoked brain potential (EP) techniques measure the specific electrical response of the brain to external sensory stimuli. With the use of computer technology as well as mathematical techniques it is possible to measure the salient features of evoked potentials which are related to sensory, perceptual, cognitive or affective responses to the evoking stimuli. The use of various evoked potential techniques can provide important information about the integrity of different brain systems.

In the last decade, the use of several evoked potential techniques has demonstrated the presence of various functional abnormalities in the brains of abstinent alcoholics (see reviews by Porjesz and Begleiter, 1981; 1983; 1985). A number of studies have shown that the Brain Stem Auditory Evoked Response (BAER) recorded in abstinent alcoholics manifests an abnormality in conduction velocity compared to those recorded in matched control subjects (Begleiter, Porjesz & Chou, 1981; Chu, Squires & Starr, 1982). Brainstem transmission time is significantly prolonged in abstinent alcoholics. With the use of different cognitive paradigms (Porjesz and Begleiter, 1979; Porjesz, Begleiter & Garrozzo, 1980; Begleiter, Porjesz & Tenner, 1980; Porjesz, Begleiter & Samuelly, 1980; Porjesz, Begleiter, Bihari & Kissin, 1987) have reported that the amplitude of the late positive component (P3) of the event-related potential (ERP) is significantly reduced in abstinent alcoholics compared to controls.

Neurophysiological deficits in sensory processes (BAER) as well as cognitive processes (P3) were originally presumed to be the consequence of alcoholism. Additional investigations examining the possible recovery of the aforementioned neurophysiological deficits uncovered differential outcome in recovery over time (Porjesz & Begleiter, 1985). Brain stem deficits were observed to recover in chronic alcoholics hospitalized over a three month period. Changes in the P3 voltage of the ERP did not show any change in abstinent alcoholics hospitalized over a similar three month period. Similar P3 amplitude deficits were observed in chronic alcoholics abstinent for 2 to 5 years.

Begleiter, Porjesz, Bihari and Kissin (1984) hypothesized that deficits in P3 voltage of the ERP may antecede the onset of chronic alcohol abuse and may be present in males at high risk for alcoholism. The investigators decided to test this hypothesis in a select group of individuals who had never had exposure to alcohol and were at high risk for alcoholism according to population genetic studies. ERPs were recorded from twenty five young sons of alcoholic fathers and twenty five control boys matched for age, socioeconomic status, school grade, and who had no family history of alcoholism. The experimental paradigm consisted of a complex visual mental rotation task to identify the orientation of a target stimulus. The high risk group showed a significantly reduced amplitude of the late positive component (P3) of the ERP. This pattern of results (low voltage P3 amplitude) is quite similar to results obtained in abstinent alcoholics (Porjesz & Begleiter, 1985), originally presumed to be solely the consequence of alcoholism. These similar findings in ERP's obtained in young, nondrinking sons of alcoholics are particularly interesting because they were obtained without administering alcohol to the subjects.

The reduced P3 voltages of the ERP obtained in young sons of alcoholics have recently been replicated in an older population (O'Connor et al., 1986). Using the identical experimental paradigm as Begleiter et al. (1984), O'Connor et al. (1986) recorded ERP's in an older (20-25 years of age) group of sons of alcoholic fathers and matched controls. None of the subjects manifested signs of problem drinking. In agreement with the observations reported by Begleiter et al. (1984), these investigators observed a significantly reduced amplitude of the P3 component of the ERP in the high risk males.

Because of the striking similarity in P3 deficits between abstinent alcoholics and males at high risk for alcoholism, Begleiter, Porjesz and Bihari (1988) used the Brainstem auditory evoked response (BAER) to assess the auditory pathway in young boys at high risk for alcoholism. The investigators examined 23 sons of alcoholics (7-13 years old) and 23 control boys matched for age, socioeconomic status and school grade. In contrast to the P3 voltage findings, no significant difference in the BAER was found between high risk and low risk boys. These results suggest that the BAER abnormalities observed in abstinent alcoholics are likely to be the consequence of alcoholism whereas the P3 deficits seen in both abstinent alcoholics and individuals at high risk for alcoholism may be antecedents of alcoholism.

In order to determine if the P3 findings in high risk individuals were modality or task specific, Begleiter, Porjesz, Rawlings and Eckardt (1987) have recently studied auditory evoked potentials in another group of high and low risk boys. An auditory oddball paradigm was developed in which subjects pressed a button to easily discriminable infrequent stimuli. In this study, all subjects were instructed to stress accuracy over speed. The subjects were 23 young boys (7-16) who were sons of alcoholic fathers and 23 control boys without a family history of alcoholism matched for age, school grade and socioeconomic status. The subjects in this study were carefully interviewed to ascertain that they had no exposure to alcohol or other illicit drugs.

It is important to note that the young sons of alcoholics tested by Begleiter et al. (1987) meet the criteria for male-limited (Type 2) alcoholism as proposed by Cloninger. All of the young high risk boys came from families in which familial alcoholism occurred only in males, was highly heritable, gave rise to severe early onset alcoholism with a high rate of recidivism requiring extensive treatment, and was accompanied by the occurrence of petty criminality. These data were obtained by conducting clinical examinations. The neurophysiological deficits observed in young male offspring of male-limited alcoholics is intriguing in light of neurochemical deficits found only in male-limited alcoholics as well as high risk individuals. These neurochemical findings will be reviewed in a subsequent section.

The results of this recent study by Begleiter et al. (1987) indicate that boys at high risk for alcoholism manifest significantly reduced amplitudes of the P3 component of the ERP. The reduced P3 voltage found in this auditory study indicates that P3 reduction in high risk males does not seem to be modality or task specific but seems to be present in the visual as well as auditory modality in easy and difficult tasks, and in speed as well as accuracy conditions.

These results of reduced P3 voltages in high risk subjects without the administration of alcohol have been replicated in three different laboratories: by O'Connor et al. (1987), by Whipple, Parker and Noble (1987) and by Steinhauer et al. (1987). As these results have now been obtained by various laboratories under different experimental conditions, they appear to be generalizable.

Other investigators have reported differences in P3 between high risk and low risk individuals only after the administration of either alcohol or placebo. Elmasian et al. (1982) studied the P3 component as well as the slow wave component of the ERP in three separate groups of subjects each consisting of 5, matched pairs (5 high risk and 5 low risk); one group served as the placebo group, the second group received a low dose of alcohol and the third group was administered a high dose of alcohol. The subjects were male college students between 20 and 25 years of age who were primarily social drinkers. The investigators observed a significant decrease in the amplitude of the P3 component in the high risk compared to the low risk subjects. However this finding was only observed after the administration of either alcohol or placebo. The investigators suggest that all subjects expected to receive alcohol; however, only high risk subjects manifest a specific expectancy for alcohol characterized by an unusual brain event. It is also suggested by the investigators that higher than normal alcohol intake in the mothers of high risk individuals might result in altered brain physiology.

Another study conducted by Neville and Schmidt (1985) examined the late positive component of the ERP between young adults at risk for alcoholism and low risk individuals. This study did not involve the ingestion of alcohol or placebo, and therefore eliminated expectancy for alcohol as a potential confounding factor. Moreover the mothers of all subjects were interviewed to determine the use of alcohol and other drugs. Group differences in the late component of the ERP were observed.

In a subsequent study Schmidt and Neville (1985) recorded ERPs in high and low risk males while they performed a visual language task. All subjects were social drinkers. The investigators found that the amplitude of the N430 component was significantly smaller in men at high risk compared to men at low risk for alcoholism. Moreover, the latency of the N430 was directly related to the amount of alcohol consumed per occasion in the high risk group. These results imply that neuronal function associated with language processes are affected by family history of alcoholism, and the interaction between family history and alcohol consumed per occasion. Taken together the neurophysiological studies conducted in populations at high risk for alcoholism indicate rather clear differences between high and low risk individuals. While many questions remain unanswered, these preliminary findings appear quite intriguing and merit further neurophysiological investigations.

NEUROCHEMISTRY:

The search for biological markers has not been restricted to neurophysiological studies but has also yielded some interesting preliminary neurochemical findings. Monoamine oxidase (MAO) is an enzyme involved in the metabolism of neurotransmitters in the central nervous system. This enzyme is also found in blood platelets where it can easily be measured. In the last decade a myriad of studies have all reported low MAO levels in alcoholics compared to controls (Gottfries et al., 1975; Oreland et al., 1983; Takahashi et al., 1976; Brown, 1977; Wiberg et al., 1977; Sullivan et al., 1979; Major & Murphy, 1978; Shaskan, 1983).

In 1980, Puchall et al. measured platelet MAO in a large group of nonalcoholic college students and their parents. The researchers observed a significant correlation between MAO in parents and offsprings. College students who manifested low levels of platelet MAO had parents who also showed low levels of MAO. It should be noted that the occurrence of alcohol abuse and alcoholism among the parents of the low MAO subjects was significantly higher than among parents of high MAO probands. These findings raise serious doubts about the possibility that low platelet MAO typically found in alcoholics are the direct result of alcohol abuse or alcoholism.

While these results are interesting it should be noted that Schuckit (1984) found no statistically significant difference in MAO level between men with a positive family history of alcoholism. The investigator measured MAO before and 180 minutes after the administration of alcohol. While statistical significance was not reached, there was a trend in the same direction, with lower MAO levels in males with a positive family history of alcoholism.

The many replications of low MAO levels in abstinent alcoholics and the demonstration that MAO levels appear to be under genetic control (Fowler et al., 1982) suggest that low MAO levels may antecede the development of alcoholism in male-limited alcoholics. A study by Alexopolous, Lieberman & Frances (1981, 1983) investigated MAO levels in alcoholic patients and their first-degree relatives. A significant relationship was found between MAO levels which were low in both alcoholics and their first degree relatives.

A recent set of observations would suggest that low MAO levels may not be prevalent in all alcoholics but may only be found in certain alcoholics. A study by von Knorring et al. (1985) indicates that two types of alcoholism may be differentiated by platelet MAO activity. The investigators studied 36 outpatient alcoholics and 34 control subjects without a family history of alcohol abuse or alcoholism. The alcoholic patients were further classified as either Type 1 (milieu-limited) or Type 2 (male-limited) alcoholics. This subclassification was accomplished on the basis of age of onset, severity of the disease, social consequences, frequency and severity of legal problems and family history. The investigators reported that platelet MAO levels were significantly lower in the Type 1 alcoholics compared to the Type 2 alcoholics. The distribution of MAO activities was essentially the same in the controls and the milieu-limited alcoholics.

A number of other studies have attempted to examine various biological variables in order to differentiate individuals at high risk for alcoholism from control subjects. Such attempts include studies of blood acetaldehyde (Schuckit & Rayses, 1979) and prolactin (Schuckit, Parker & Rossman, 1983). The lack of experimental replication for the aforementioned biochemical studies do not make these variables viable candidates as potential biological markers. At present there is no compelling evidence to indicate that rates of alcohol metabolism are associated with the risk for alcoholism (Utne et al., 1977; Schuckit, 1981).

CONCLUDING REMARKS:

It is quite obvious that in the last five years there have been some significant research developments in the study of individuals at high risk for alcoholism. A number of biological as well as behavioral variables have already been reported to differentiate subjects at high risk for alcoholism from individuals at low risk. However, it is at present totally unknown whether the myriad of variables which have already been identified are indeed predictive of subsequent alcohol abuse or alcoholism. Prospective longitudinal research is absolutely essential in order to evaluate the validity, reliability and predictability of those variables as potential phenotypic markers for a predisposition toward alcoholism. Those longitudinal studies can be considered high risk for the investigator unless a set of well delineated variables with high potential can be culled for longitudinal studies.

The following criteria are necessary for the identification of a potential biological marker:

1. Studies of subjects from the general population should show that the trait
    a) can be reliably measured and is stable over time (Table 1)
    b) is genetically transmitted (Table 2)
    c) the "abnormal" trait has a low base rate (Table 3)
    d) identifies individuals at risk (Table 4).
2. Studies in patients should show that the trait
    a) is prevalent in the patient population (Table 5)
    b) is present during symptom remission (Table 6)
    c) occurs among the first degree relatives of the index case at a rate higher than that of the normal population (Table 7)
    d) segregates with the illness in affected relatives (Table 8).

Tables 1-4 indicate results of neurophysiological tests (EEG, sensory EPs and long latency ERPs) of subjects in the general population for each of these criteria, respectively. The criteria for the identification of a potential phenotypic marker are only discussed within the context of neurophysiological experiments, as they provide the most compelling data to qualify as biological markers.

INSERT TABLES 1, 2, 3 and 4 ABOUT HERE

As can be seen in each of these tables, neurophysiological measures appear very promising in terms of the above criteria.

Tables 5-8 indicate results of neurophysiological tests in patients for each of the criteria of a genetic marker, respectively.

INSERT TABLES 5, 6, 7 and 8 ABOUT HERE

As these tables indicate, neurophysiological measures meet the criteria for the identification of a marker in most of these patient-related criteria except those that have not yet been examined. Studies are underway to investigate whether neurophysiological measures segregate with the illness in the affected relatives. Thus, on the basis of currently available data, it appears that neurophysiological measures are likely candidates as potential markers of alcoholism.

Future studies must of necessity heed recent data which indicate that alcoholism is a heterogeneous disease with multiple clinical types of alcohol abuse. Studies of high risk populations should obtain detailed clinical characterization of alcoholism in the family, since it is now established that different types of alcoholism may be corrrelated with different gene-environment interactions. Investigators should no longer focus on statistical techniques which attempt to compare group means from high risk versus low risk populations. More attention needs to be focused on outliers as well as the presence of different distributions or populations within the high risk group. Studies of subjects at high risk for alcoholism should not only focus on abstainers or mild drinkers, but should include drinkers as well for possible comparisons.

At present, studies on high risk populations have been limited to male subjects. There is need to investigate daughters of alcoholics and to compare them to sons of alcoholics. Moreover, there is need to assess the relationship between various potential markers and the pattern of incidence of alcoholism in first and second degree relatives.

It is becoming increasingly obvious that the development of alcoholism is not likely to be the result of a single biological or behavioral factor. Indeed the disease of alcoholism is likely to reflect the complex interactions of biological and behavioral predisposing factors in conjunction with environmental precipitating factors. Therefore it is incumbent upon future investigators to evaluate biological as well as behavioral variables, simultaneously, and to assess their interactions.

While studies involving young individuals at high risk for alcoholism cannot examine the pharmacogenetics of alcohol, the effects of alcohol should be evaluated in older subjects at high risk for alcoholism. Initial sensitivity , tolerance, as well as the reinforcing properties of alcohol should be studied in populations at high risk. Biological and behavioral studies of high risk populations should lead to the development of valid and reliable phenotypic markers which should eventually be used in family studies in conjunction with molecular genetics.

The identification of a potential biological marker or predictor would be of substantial help in our understanding of etiological factors in alcoholism. Biological markers for alcoholism represent one of the most promising areas of research, because unlike other psychopathopharmacological agent with the endogenous physiological and biochemical processes.

The identification of subjects vulnerable for the future development of alcoholism is critical to the development of a primary prevention effort. The first step to be undertaken in the implementation of rational primary prevention is the identification of vulnerability factors in subjects at high risk for alcoholism. The data obtained from such studies may lead to a better understanding of etiological factors in alcoholism and subsequently the development of appropriate treatment regimens. The identification of vulnerable individuals and the possible elucidation of some etiological factors are crucial in focusing prevention initiatives and resources.

TABLE 1

MARKER CAN BE RELIABLY MEASURED AND IS STABLE OVER TIME

TEST-RETEST CORRELATIONS:	6 MONTHS - 18 MONTHS
EEG (frequency analysis)	.62 - .87
Short latency EP (Sensory)	.81 - .96
Long latency EP (Cognitive)	.79 - .93

TABLE 2

THE MARKER SHOULD BE GENETICALLY TRANSMITTED

	MZ	DZ
EEG (All frequencies)	.61 - .79	.23 - .34
EEG (Alpha only)	.69 - .81	.31 - .37
Early EP (Sensory)	.78 - .86	.34 - .35
Late EP (Cognitive)	.64 - .81	- .32 - .38

TABLE 3

THE MARKER (ABNORMAL) MANIFESTATION SHOULD HAVE A LOW BASE RATE IN NORMAL POPULATION

EEG (All frequencies)	Abnormal less than 5%
Early EP (Sensory)	Abnormal less than 4% (?)
Late EP (Cognitive)	Abnormal less than 4% (?)

TABLE 4

THE MARKER SHOULD IDENTIFY INDIVIDUALS AT RISK

EEG	Current data on correct identification not available
Early EP (Sensory)	Does not appear useful to identify individuals at risk
Late EP (Cognitive)	35% of sons of all alcoholic fathers
	87% of sons of Type 2 alcoholic fathers

TABLE 5

THE MARKER SHOULD BE PREVALENT IN THE PATIENT POPULATION

EEG abnormalities	Approximately 55% of short-term abstinent alcoholics.
Early EP (Sensory)	Approximately 61% of short-term abstinent alcoholics.
Late EP (Cognitive)	Approximately 74% of short-term abstinent alcoholics.

TABLE 6

THE MARKER SHOULD BE PRESENT DURING SYMPTOM REMISSION

EEG abnormalities	Approximately 19% of long-term abstinent alcoholics.
Early EP (Sensory)	Approximately 7% of long-term abstinent alcoholics.
Late EP (Cognitive)	Approximately 43% of long-term abstinent alcoholics.

TABLE 7

THE MARKER SHOULD OCCUR AMONG FIRST DEGREE RELATIVES OF PATIENTS AT A RATE HIGHER THAN THAT OF "NORMAL" POPULATION

EEG:	Significantly higher incidence of EEG abnormalities in high risk individuals compared to control subjects.
Early EP (Sensory)	Not available.
Late EP (Cognitive)	Significantly higher incidence of late EP abnormalities in first degree relatives of patients compared to normal population.

TABLE 8

THE MARKER SHOULD SEGREGATE WITH THE ILLNESS IN AFFECTED RELATIVES

EEG	Data not available.
Early EP (Sensory)	Data not available.
Late EP (Cognitive)	Only preliminary data available.

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NEUROBEHAVIORAL AND GENETIC BLOOD MARKERS FOR ALCOHOLISM RISK

TESTIMONY
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SENATOR JOHN GLENN, CHIARMAN
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NEUROBEHAVIORAL MARKERS OF ALCOHOLISM RISK:

A number of psychiatric disorders have been found to be associated with neurological abnormalities. Twenty five years of research has now firmly established an excess prevalence of neurological abnormalities among schizophrenic patients though the etiology of the neurological problems is still uncertain. Unlike schizophrenia, the search for neurobehavioral correlates in alcoholism is relatively new. Until relatively recently it had been common to study the etiology of alcoholism by examining alcoholics who obviously had the diathesis for the disorder, but who also had experienced the deleterious effects of drinking alcohol for many years. It was reasonable, then, to conclude that any deficits seen in neurobehavioral functioning was due to the alcohol consumed. Fortunately, about five years ago investigators began to ask whether some of the deficits in neurobehavioral functioning might have existed before the onset of the problem and might be prodromal in some way.

Static Ataxia: Evidence has now accumulated suggesting that neurological abnormalities may exist in high risk individuals. These abnormalities are not generally so gross as to impair normal functioning but are readily observable under standardized laboratory conditions. One neurobehavioral function which has been of particular interest is that of static ataxia. Static ataxia refers simply to ataxia at rest, or incoordination of balance or equilibrium.

In our first investigation of this phenomenon, children of alcoholics, normal controls and children of depressives were found to differ on static ataxia. The children of the alcoholics swayed more than the normal control children when asked to stand as steady as possible while we measured their sway with a mechanical device that quantifies the child's movement. Of interest was the fact that the children of fathers who met criteria for major depressive disorder did not differ from the control children, suggesting to us that the deficit seen was specific to alcoholism and not just a result of having inherited a tendency for psychiatric problems nor due to non-specific environmental effects such as living with a parent with either alcoholism or depression. These results were quite encouraging because they suggest a specificity of this marker for alcoholism.

More recent work in our laboratory included children of male alcoholics from high density families who were compared on two measures of static ataxia with children of controls. By way of note, high density families are those in which alcoholism occurs frequently within the pedigrees of the identified index case. We reasoned that if alcoholism has a genetic basis, then starting with families in which there are multiple cases of the disorder might increase the likelihood of uncovering markers for this genetic predisposition. We selected families in which there were a minimum of two alcoholic brothers. Selecting families through a pair of brothers enabled us to identify pedigrees that were clearly transmitting the disorder across generations.

When the children from these high density families were compared with children from control families in which no relatives were alcoholic, we were able to confirm the earlier reports of two other laboratories as well as our own in which children of alcoholics were found to sway more than control children. Our most recent findings extend the observation to include children of alcoholics who were, because of the design of the larger study, also the nieces and nephews of alcoholics. Many had grandparents and/or great aunts and uncles who were alcoholic as well. Analysis of the extended pedigrees indicated an average of 3.3 males and 0.33 female first- or second-degree relatives were alcoholic. Thus, it would appear that the subtype of alcoholism exhibited in fathers of the high risk children assessed here is a more genetically mediated form sometimes referred to as "type 2" or "male-limited" after the observations of Cloninger and colleagues in St. Louis. Our results suggest that sway may be a useful psychobiological marker, particularly in families in which there is a heavy loading of this more genetically mediated form of alcoholism.

Event-Related Potential Characteristics: There is an extensive literature linking chronic alcoholism to cognitive dysfunction using both neuropsychological and neurophysiological measures. A significant breakthrough in understanding the etiology of alcoholism occurred when it was established that children of alcoholics, who are at increased risk for developing alcoholism, differ from children of nonalcoholic individuals in both cognitive functioning and electrophysiological activity.

Further evidence in support of a genetic contribution to neuroelectric activity, which in turn appears to differentiate those at high and low risk for developing alcoholism, comes from a variety of sources including studies examining EEG alpha activity and event-related potentials (ERPs), particularly the P300 component of the ERP. P300 is a positive shift at the scalp which occurs with a latency of approximately 300 msec. If alterations in cognitive functioning and electrophysiological activity are seen in relatives of alcoholics who are not themselves alcoholic, then it is possible that some of the cognitive capacity and electrophysiological activity that distinguishes alcoholics from nonalcoholics is, in fact, antecedent to the development of alcoholism. Indicators or markers from these two domains could then be considered candidates as "vulnerability" markers rather than "episode" markers -- that is, markers for risk as contrasted with markers for alcohol consumption per se.

We found that variations in P300 are apparent in the adult siblings and parents of alcoholic individuals as well as their children. A number of previous investigations have linked P300 to risk for developing alcoholism. Among the most impressive demonstrations of this are those seen in children. We have found that it is latency of P300 that differentiates adult siblings of alcoholics, all of the siblings having longer latency than control subjects.

Children, in contrast, show a reduction in amplitude, suggesting that developmental considerations may have some bearing on the expression of "marker" characteristics. With a larger sample and employing an analysis that includes the fathers and mothers of the adult proband alcoholic, it now appears that within these families with a high genetic loading, the nonalcoholic sibling differs from all of his first degree relatives in having a shorter latency of P300.

Minor children have also been tested who are the offspring of the alcoholics and their siblings, in addition to children of normal controls. Comparison of event-related potentials in these children of alcoholics and controls revealed two major findings: an enhancement of frontal negativity in the children of alcoholics and an overall reduction in P300 amplitude among the high risk children.

The maximum amplitude of another ERP component (N250) typically occurs at the vertex electrode in adults. However, in children, N250 is most prominent over the frontal portion of the brain. This negativity over the frontal region appears to decline with age so that adolescents show less negativity than younger children and older adults are less negative than adolescents. This frontal negativity has been viewed as being associated with the development of more posterior (parietal) Slow Wave, and its decrease has been suggested as reflecting maturational development. Consequently, the findings for greater negativity in the children of alcoholics in the present study suggest an interesting hypothesis: that the high risk children may be showing an electrocortical sign of maturational lag. Further research is needed with groups at various ages to document this observation. Thus, positive findings are consistently found for minor children including the work of Begleiter and colleagues, and our own. In addition, we have found differences in ERP characteristics of adult men from high risk families when contrasted with low risk families.

GENETIC BLOOD MARKERS:

The most convincing evidence that alcoholism is transmitted genetically, other than demonstrating a specific biomedical gene product, would be to demonstrate genetic linkage of an alcoholism susceptibility (AS) gene (or genes) to a well-defined polymorphic marker. Polymorphic markers are those that commonly exist in the population in two or more discrete forms. For a potential marker to be most highly informative, it should be reliably detectable as well as independent of the disease state, that is, it is unaffected by whether or not the at risk individual is currently affected by the illness. Ideally, it should be localized to a particular chromosomal region.

A number of polymorphisms in man have been found in blood, making it possible to attempt to find genetic linkage or association between alcoholism and these markers. Most studies have concentrated on only two markers (ABO or ABH secretor systems), though two laboratories, our own and one other, have considered other markers located on various chromosomes. Association involves showing that the frequency of a particular allele differs in a population of affected individuals from that seen in a normal control population. Linkage assesses the co-segregation of a particular disorder within families with a marker. If linkage is demonstrated, it is assumed that the marker -- in this case a particular blood group -- is on the same chromosome as the AS gene and is in sufficient proximity to the marker so that the loci fail to follow Mendel's law of random assortment. (It is possible to have genetic linkage between an AS gene and a marker without there being association between those loci.)

We investigated the possibility of genetic linkage between the AS gene and six blood group markers: Rhesus and Duffy on chromosome 1, MNS on chromosome 4, ABO on chromosome 9, Kidd on chromosome 18 and Kell (unassigned), among 30 nuclear families chosen because there was a pair of alcoholic brothers present. Only those families with a demonstrated absence of other psychiatric disorders in first and second degree relatives (e.g., affective disorders, schizophrenia) were included. Personal psychiatric interviews were conducted for all family members who participated in blood typing (N = 124) and who were first degree relatives, allowing for determination of whether or not the subject met accepted criteria for alcoholism. All probands and affected siblings included in the present analysis met these criteria. Family history information was sought for second and third degree relatives of the proband and those first degree relatives who were deceased, or did not participate in the face-to-face interview and blood draw. In this way, pedigrees were extended to include a total of 220 first degree, 348 second degree and 288 third degree relatives. This information was used to complete a segregation analysis to determine a suitable genetic model to use for the linkage analysis.

Evidence for genetic linkage between the MNS blood group and a putative alcoholism susceptibility gene was found using two methods to evaluate the genetic linkage: sib-pair analysis which involves comparing similarity of marker status in pairs of siblings and using a computer program which tests linkage quantitatively (LIPED).

Further study is required to firmly establish both the genetic model for the alcoholism susceptibility gene and the genetic linkage between this gene and the MNS blood group. As part of a larger study, a number of high density pedigrees are being ascertained. Therefore, we are currently collecting more families to determine if this finding can be replicated.

NEUROBIOLOGICAL AND PSYCHOSOCIAL DETERMINANTS OF ALCOHOL DRINKING

PREPARED FOR
THE UNITED STATES SENATE
COMMITTEE ON GOVERNMENTAL AFFAIRS
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INITIATION AND MAINTENANCE OF SOCIAL DRINKING

Factors associated with initiation and maintenance of social drinking may be quite different from those which lead to abusive drinking or alcoholism. In order to provide a framework for understanding addiction to alcohol, it may be useful to provide background on how the consumption of alcohol relates to other ingestive behavior both in animals and in man.

The expression of any voluntary human behavior is the result of a variety of factors that interact to determine for any given time whether or not a particular behavior will emerge. Consumption of alcohol can be considered an appetitive behavior similar to the drive for food. The type of food consumed, as well as its rate and pattern of ingestion, are quite variable among individuals even within the same species. Animal studies concerned with ingestive behavior have uncovered critical brain structures (e.g., lateral and ventromedial nuclei of the hypothalamus) that regulate both the initiation, timing and ultimately the satiety for food. These structures are part of the "wired in" or neuroanatomical features of the organism that are determined by its genetic endowment. Of course, these neuroanatomical structures function in the way they do because of a complex array of neurochemical and neurophysiological events that take place within any one of these functionally disparate units.

When ingestive behavior is considered in the human, a host of other environmental factors come into play. In the case of alcohol, these include the person's socioeconomic status, his or her cultural milieu, religious orientation, if any, ethnic background and the time in which he or she was born. Similar influences can be seen in the choice of food the individual consumes. Here too, secular trends are evident in the move away from high calorie, high cholesterol diets, so common at the turn of century when many of our ancestors were farmers engaged in physical labor, to those diets associated with youth, health and slimness. For the drinking of alcohol, we see changes across time so that the age of onset of alcoholism is younger with each decade of birth (Reich et al., in press).

To this list of non-familial environmental factors that influence food or alcohol consumption, one must add familial environmental factors which have been shown to affect drinking. Clearly the amount of drinking as well as the occasions upon which it is consumed within the family unit influence other members' drinking. In some families, heavy drinking is so commonplace that it becomes the subcultural norm within the family. Similarly, in some homes alcohol is consumed only in the case of religious celebrations or on special occasions, such as holidays. In these families, use of alcohol to alleviate stress or as a behavior in its own right (alcohol seeking) has not been exhibited by adult members, so that children will not have learned these connections from salient role models by the time they reach the legal drinking age.

Thus, the initiation of alcohol drinking, like eating, is determined by a host of factors, all of which probably interact in varying degrees at various times. If alcohol by its very nature is more addictive than food, then understanding how drinking is maintained at non-abusive and non-addictive levels is the real challenge. Stated another way, all of us may be capable of being passively addicted to alcohol at the neuropharmacological level. For example, when laboratory rats are forced to drink alcohol/water solutions as their sole fluid source for a number of days then are suddenly withdrawn from the alcohol, many will go into tonic-clonic seizures. Left to its own devices, however, the laboratory rat generally avoids alcohol unless he is either fooled into believing the alcohol is something else (e.g., sugar) through adulteration with saccharine or sucrose, has nothing else to drink, or belongs to a strain of rats inbred for their tendency to drink alcohol. What, then, prevents the typical laboratory rat, or human, from over imbibing? We do not know the answer to this question. However, we have begun to find "markers" or "pointers" associated with human overindulgence that will ultimately lead us to an understanding of the underlying neurobehavioral predisposition we call vulnerability to alcoholism.

SCIENTIFIC MODELS OF ETIOLOGY - ABUSIVE AND ALCOHOLIC DRINKING

The factors which operate to initiate drinking may not be the same ones that lead to abusive or problem drinking. Recognizing the multifactorial etiology of abusive drinking, we have developed a vulnerability model for understanding why some individuals can drink throughout their lives at moderate levels without becoming addicted while others cannot (Hill et al., 1987). Multifactorial models have not been common in the alcohol field, with various groups claiming the supremacy of one etiological model over another. For convenience, we have grouped these models into broad categories (ecological, developmental, and genetic). Each of these general models has submodels that particular investigators tend to espouse. Some of the major submodels will be reviewed to provide a broader context for the vulnerability model which we have developed and which we are currently testing.

THE ECOLOGICAL MODEL

Limiting Availability: Development of alcohol problems can be affected by both the supply of alcohol as well as the demand for it. Those concerned with the supply side have emphasized that the availability of alcohol and legal proscriptions for its use are predictive of alcohol problems in a society. Among the variables which the Institute of Medicine (1980) has identified as affecting the availability of alcohol in the community are licensing restrictions, taxation, number of outlets, age and hour limits, and the enforcement of relevant laws.

Family Interaction Patterns: This view holds that whether or not a family transmits alcoholism to the next generation is determined by how the family deals with the presence of an alcoholic individual in the family. Interestingly, those families that are able to maintain family rituals without disruption from the alcoholic individual maintain an identity that protects them from transmitting the problem to the next generation more than do families in which the alcoholic's behavior does disrupt holidays and other rituals (Wolin et al., 1980) forcing them to loose all family identity beyond that related to its pathology. Family experiences include patterns of social interaction. Jacob (1987) has noted that family interaction patterns of alcoholics are characterized by greater negative interaction with lesser amounts of constructive criticism, which alcohol serves to accentuate. Thus, through poor family communication patterns, children experience many disadvantages, some of which may be related to their increased chance for later development of alcohol problems.

THE DEVELOPMENTAL MODEL

Social Learning Theory: Under the developmental heading belongs those experimental factors that appear to operate in the life of the child to influence his or her outcome with regard to drinking. In this model, the premises of social learning theory are influential, predicting that an individual learns behavior, whether normative or deviant, through modeling the behavior of others (Bandura, 1969). From this theory one can predict that an individual will learn to become alcoholic by modeling the behaviors of alcoholics through a system of beliefs about the effects that alcohol has on functioning.

Learning any behavior usually requires numerous trials to reach the desired end, to which any one can attest who has had to memorize a list of words from a foreign language. Yet, some instances of learning take place in a single trial, either because they are so particularly noxious or because the emotional overtones are so great. Most of us have learned to associate an event with a time and place, often retaining that memory for a lifetime if the emotional relevance of the event was particularly salient. Many people remember where they were and by whom they were told that John Kennedy had been shot. Bandura (1969) speaks of "no trial" learning in which new behaviors are acquired through modeling. If the model has particular reward value, as parents certainly do given their early ties to the emotional rewards of the child, learning particular behaviors is facilitated. Further, in observing the parent's style of coping, the child learns to adopt similar modes of coping, albeit maladaptive in the case of the child who emulates the alcoholic parent, and becomes alcoholic too.

Personality Theory: Etiological theories of alcoholism that find support from observations of personality variants among alcoholics and their high risk relatives will be discussed under developmental theories of etiology. While there is clear evidence that particular personality variants are heritable (Gottesman, 1963), there is evidence that these traits appear to be modified by the environment to a varying degree. Personality is a complex behavioral trait that is most probably determined by polygenetic influences. This is not surprising when one considers that most gene-determined human variability is derived from the simultaneous expression of many genes, each of which contributes only a minor effect upon the trait in question, but when acting in concert determines an emerging trait such as skin color. Because traits that are polygenic in origin appear to be maximally sensitive to environmental factors, it is reasonable to assume that the enduring traits seen over time in particular individuals are the result of both hereditary predisposition and developmental experiences that shape the personality.

As will be seen in a later discussion of the vulnerability model, personality is considered to be a premorbid characteristic of the individual that is orthogonal to symptomatic aspects of alcoholism such that it does not "cause" alcoholism but is one expression of the underlying neurobiological functioning of the individual. In this view, both personality and cognitive factors (e.g., memory, learning, motivation and other information processing characteristics) interact to determine the response bias of the individual to stimuli in general and to alcohol in particular. Personality variants were not always accorded this role in the etiology of alcoholism. In fact, the "addictive" personality at one time was considered the primary "cause" of alcoholism and substance abuse. This view not only attributed causality to personality, but did so through a univariate explanation -- one single personality type. This view probably emerged from psychoanalytic theory which viewed the alcoholic as orally fixated.

We now realize the inadequacy of any theory which describes alcoholism from a single trait perspective. Alcoholism is now known to be a heterogeneous disorder. Analysis of clinical samples has revealed subtypes differing in the degree to which environmental factors determine whether or not the individual will become alcoholic (Cloninger et al., 1981). Heterogeneity has also been demonstrated when personality assessments are used in alcoholic samples. Two subtypes have been identified using the Minnesota Multiphasic Personality Inventory (MMPI) that have been replicated across a number of studies (Morey and Blashfield, 1981; Skinner and Allen, 1982). These are the passive-aggressive sociopathic subtypes and the chronically-distressed neurotic subtypes, which can further be distinguished by their drinking styles and resulting problems associated with drinking. The neurotic drinkers tend to be heavier drinkers with more problems than the sociopathic subtype who drinks more moderately and with fewer detrimental consequences (Skinner and Allen, 1982).

The drinking of alcohol over a period of time can have consequences for both cognitive functioning and personality expression. Therefore, among the most informative studies of personality as a possible diathesis marker are those which have examined personality in the same individual both before and after the onset of the alcoholism, or those studies that have looked at high risk relatives of alcoholics who were not themselves alcoholic. A few longitudinal studies have been completed in which persons who later became alcoholic were assessed before the confounding effects of alcohol were manifest. In one study, MMPI profiles of alcoholics undergoing treatment were compared with profiles obtained for the same individuals while in college, before they became alcoholic (Hoffman et al., 1974). Next, they compared their college profiles with those of their classmates who did not later become alcoholic. Two scales of the MMPI were found to be elevated in the prealcoholics: Scale 4 (Psychopathic Deviate scale) and Scale 9 (Hypomania scale), suggesting that the prealcoholic college student tended to be more gregarious, impulsive and nonconforming than his peers who did not become alcoholic.

While no significant elevations were noted on MMPI scales indicative of subjective distress among these prealcoholic individuals, once they became alcoholic their MMPI profiles indicated elevations on both the Psychasthenia and Depression scales, as well as continued elevations on the Psychopathic Deviate scale. This underscores the importance of not drawing conclusions about personality variants as markers for risk through the use of clinically diagnosed alcoholics alone. Longitudinal studies of high risk individuals would be expected to give the greatest pay-off in this regard, followed by a cross-sectional look at high risk relatives of alcoholics who are not themselves alcoholic.

Currently, we are engaged in a study of cognitive and personality factors in relatives of alcoholics that seeks to identify risk markers from a variety of domains, including personality dimensions. Having administered personality inventories to alcoholic probands, their alcoholic and nonalcoholic brothers and their parents, we were in a position to determine which of the reported elevation of personality scales were a consequence of drinking and which were possibly vulnerability markers. Like others, we found significant elevations in clinical scales when we compared the group of alcoholic probands with control subjects. In fact, five scales were found to be significantly elevated: Psychopathic Deviate (Pd), Mania (Ma), Psychasthenia (Pt), Schizophrenia (Sc), and Paranoia (Pa). However, when we contrasted the nonalcoholic brothers of these alcoholics with the control group, only two of these differences remained (Pd and Sc), suggesting that these characteristics might be vulnerability markers. Because of the convergence between our data and Hoffman et al. (1974), we are inclined to be more confident about the Pd finding.

THE GENETIC MODEL

The genetic model is probably the most developed, drawing on a variety of methodologies that have yielded results suggesting a genetic propensity to develop alcoholism. Although this propensity may not be the necessary and sufficient condition for the development of alcohol-related problems, it may be the necessary condition. This might appear to overstate the case for genetics, given that some alcoholics appear to have a non-familial form (no first-degree biological relatives with alcoholism). However, not all individuals who carry a genotype express it so that some apparently nongenetic types may, in fact, be so.

Increased Risk in First Degree Relatives: Evidence for the notion that vulnerability for developing alcoholism is genetically mediated comes from a number of sources. Alcoholism clearly runs in families with one review noting over 100 studies showing that alcoholism is three to five times higher among the first-degree relatives of alcoholics than it is in the general population (Cotton, 1979).

A Single Addiction-Prone Predisposition? Alcoholism, like many other disorders, appears to run "true to type." While poly-drug abuse appears to have been common since about 1970, individuals can be found who abuse only one type of drug. These single drug abusers tend to be clustered among either alcoholics or heroin abusers more than among other types of drug use. Particularly among older alcoholics, one can find almost a disdain for illicit drugs.

Using samples of "pure" alcoholics and "pure" heroin abusers, we were able to follow the transmission patterns of abuse across generations, finding that the risk for alcoholism among alcoholic probands was greater among their siblings than among siblings of heroin abusers (Hill et al., 1977). This type of result had also been found in Germany in 1933 by Polich who investigated the family histories of alcoholics and opiate addicts finding alcoholism in 22% of the brothers of alcoholics and in 6% of the brothers of opiate addicts. Whereas alcoholism occurred in 47% of the fathers of alcoholics, alcoholism occurred in only 6% of the fathers of the opiate addicts. These results from two countries at different points in time suggest a "true to type" inheritance of abuse.

These results could not be explained on the basis of differential accessibility to the two drugs for two reasons. First, the St. Louis sample was drawn from the inner city and consisted primarily of Black subjects who told us they had easy access to all drugs at the time the study was done. Secondly, by examining a third group that abused both heroin and alcohol predominantly, we were able to determine which type of abuse came first in their life histories. Interestingly, the family histories of these individuals (whether alcohol abusing or heroin abusing relatives were present) predict which abuse came first. Those with alcohol abusing relatives tended to become addicted to alcohol first and heroin later. Those with heroin abusing relatives tended to become addicted to heroin first and alcohol later.

Familial transmission of alcoholism and opiate dependence was tested using the independent model, the most general form of the multifactorial model of disease transmission. Specifically, all possible correlations between relatives and probands (opiate abusing or alcoholic) are calculated. From this, the phenotypic correlation is calculated. If the familial factors relevant to the two disorders are uncorrelated or independent, then the expected phenotypic correlation will be zero. If the familial factors relevant to the two disorders are the same, then the expected correlation is 1. If the two disorders share some, but not all, common etiological factors, then the expected phenotypic correlation will be less than 1 but greater than 0. Results of the analysis revealed a correlation of 0.00 - 0.25, indicating little overall familial aggregation for the two disorders.

Evidence from Animal Studies: Having identified this process in clinical samples, we undertook a study of this question using laboratory rats inbred for their ability to solve mazes: the Tryon Maze Bright and Tryon Maze Dull animals. Our goal was to compare the intake of alcohol and morphine solutions in these two strains. These strains were interesting because of differences in exploratory activity. The Maze Dull animal is less fearful in open spaces and therefore traverses more area in a given time than does his Maze Bright counterpart who is more fearful in open spaces, cowering in the corner or passively alert, searching for any cue that danger might be lurking. If one assumes that animals that are less fearful and engage in more exploration may be more likely to engage in appetitive behavior, then it would be predicted that the Maze Dull (S3) animals should show greater intake of both morphine or alcohol solutions than their Maze Bright (S1) counterparts. In fact, the strain that drank significantly more of the morphine solution, the S3 strain, was not the strain that drank significantly more alcohol. Rather the S1 strain drank more alcohol solution than did the S3 strain. These results confirmed in laboratory animals the independent transmission of preference for these two different types of drugs. Both studies strongly suggest that there is not a single genetic vulnerability to drug abuse, but rather that alcohol abuse and alcoholism is a separate disorder from that seen in heroin abuse and quite possibly other drug abuse disorders (e.g., cocaine abuse).

Twin studies: Although not a marker as such, concordance in drinking behavior among genetically related individuals should follow some predictable pattern if drinking behavior has a genetic basis. The evidence suggests a fairly high concordance. Kaij (1960) studied 174 Swedish twin pairs selected because one member of the pair had appeared on the national register of alcohol abusers. Using a five-point scale from abstention to chronic alcoholism, he found about twice the concordance in monozygotic twins (Mz) (53%) as in dizygotic twins (Dz) (28%) in terms of having the same grade of drinking (1-5). He also found greater concordance when the chronically alcoholic proband was a member of a monozygotic twin pair (70%) than a member of a dizygotic pair (32%). Partanen et al. (1966) in Finland failed to find greater concordance in monozygotic than dizygotic twins for "out of control" drinking or "consequences of drinking." These twins were drawn from a general population sample, whereas Kaij's sample was chosen from the registry of alcohol abusers. However, in both frequency and quantity per occasion, there was greater concordance in the monozygotics than the dizygotics Partanen studied, suggesting a genetic influence in drinking. Heritability estimates were 0.39 for frequency and 0.36 for quantity per occasion.

Two other twin studies are worthy of mention. One was a sample of twins gleaned from among the 600,000 juniors who took the National Merit Scholarship

Questionnaire Test (Loehlin, 1972). Greater concordance was found among Mz twins than among Dz twins for questions related to heavy drinking among the 850 twins identified in this sample. Jonsson and Nilsson (1968), in their study of 1500 Swedish twin pairs, also found greater concordance among the Mz twins than the Dz twins in the quantity consumed.

All of these findings taken together suggest that genetic influences operate to make concordance of drinking patterns more similar among relatives, with those sharing the greater proportion of their genes even more similar.

Adoption Studies: Adoption studies indicate that risk for developing alcoholism is related to the biological parent rather than the foster parent (Goodwin et al., 1973; Cadoret and Gath, 1978; Bohman, 1978). Schuckit et al. (1972), studying the half sibs of alcoholic probands, demonstrated that alcoholism could be transmitted from parent to offspring without the necessity of being raised by the alcoholic. This observation, as well as findings from the Danish adoption study (Goodwin et al., 1973), established that transmission of alcoholism from father to son can occur even if the child is given up for adoption and removed from the alcoholic parent. Indeed, further studies on this population showed that even when the sons of alcoholics were raised with their alcoholic fathers, their risk for developing alcoholism was no greater than when they were removed from their alcoholic fathers.

A replication by Bohman (1978), showing that adopted away offspring of alcoholics have significantly increased risk, makes the Goodwin finding even more certain. The reanalysis of Bohman's Swedish adoption data has led Cloninger et al. (1981) to propose that there are two genetically distinct forms of alcoholism. In the first, "milieu-limited form," the disorder can be characterized by mild alcohol abuse in parents, minimal criminality, and strong environmental influence on the frequency and severity of alcoholism. In the second case, the "male-limited form" which is transmitted almost entirely from father to son, the disorder is characterized by an association with severe criminality and is minimally influenced by environmental factors. The latter form most probably is typical of the type seen in the Danish adoption series.

WHAT IS BEING TRANSMITTED? THE SEARCH FOR MARKERS FOR ALCOHOLISM RISK

The Heterogeneity Problem: The significance of studies like those of Cloninger et al. (1981) is, firstly, that genetic influences have now been firmly established and secondly, that the heterogeneity of the disorder has been, to some degree, characterized. Although there may be many subtypes of alcoholism not yet delineated, recognition of the male-limited and milieu-limited forms has facilitated identification of biological markers for alcoholism. This heterogeneity, exemplified by these two subtypes varying in the degree to which gene-environment interactions occur, indicates that environmental factors must be taken into account in any analysis that seeks to find indicators correlated closely with the underlying liability distribution.

Among the most promising markers are those which can be grouped as metabolic, behavioral, neurobehavioral and chromosomal. Because our own research has concentrated on the neurobehavioral and chromosomal markers, only brief mention will be made of metabolic and behavioral markers.

Metabolic Markers: Metabolic markers are of interest because of their association with particular populations who appear to show protection from alcoholism. Orientals have traditionally exhibited lower rates of alcoholism than Caucasians. Therefore, it was reasonable to search for possible biological differences among the Orientals. Alcohol is oxidized to acetaldehyde by the enzyme alcohol dehydrogenase (ADH). Particular genetic variants of this enzyme are associated with particular polypeptides each of which has different enzyme activity. One form which has higher enzymatic activity is far more common among Orientals than Caucasians. The higher enzymatic activity of this "atypical" isoenzyme could increase elimination rates and lead to production of greater amounts of acetaldehyde which may be associated with physiological discomfort. Orientals are also known to have an exaggerated behavioral response to alcohol ingestion which includes facial flushing, muscle weakness, dizziness, tachycardia and hypotension.

Behavioral Markers: One line of research has centered on asking whether or not persons who are at high risk for developing alcohol problems might experience the effects of alcohol differently than those who are not. Schuckit et al. (1972; 1980), in a series of studies, examined the effects of administered alcohol on subjects' mood both before and after drinking. Subjects had been selected from among a pool of college students, some of whom had a family history of alcoholism and some of whom did not. These studies indicated that the students with a family history responded to alcohol differently than those without an alcoholic relative. Those with a higher risk for developing alcoholism responded by rating themselves lower on a global rating scale of how "high" they felt. These findings suggest that the effect that alcohol has on brain areas associated with emotional regulation may be different in people who have a higher risk for developing problems. If the high risk subjects experience less of a high at the same dose of alcohol than do their counterparts, it may indicate that these individuals would need to drink more alcohol to get the same effect, thereby increasing their chances of becoming physically dependent on it.

Neurobehavioral Markers: A number of psychiatric disorders have been found to be associated with neurological abnormalities (Woods et al., 1986). Schizophrenia has been one of the most widely studied. Twenty five years of research has now firmly established an excess prevalence of neurological abnormalities among schizophrenic patients. In spite of the long history of research in this area, interpretation of the results remains unclear. The etiology of the neurological problems is uncertain, though there is evidence of an increased prevalence of obstetric complications among schizophrenics. Unlike schizophrenia, the search for neurobehavioral correlates in alcoholism is relatively new. Until relatively recently it had been common to study the etiology of alcoholism by examining alcoholics who obviously had the diathesis for the disorder, but who also had experienced the deleterious effects of drinking alcohol for many years. It was reasonable, then, to conclude that any deficits seen in neurobehavioral functioning was due to the alcohol consumed. Fortunately, about five years ago investigators began to ask whether some of the deficits in neurobehavioral functioning might have existed before the onset of the problem and might be prodromal in some way.

Neurological Abnormalities - Static Ataxia: There is now accumulated evidence suggesting that neurological abnormalities may exist in high risk individuals. These abnormalities are not generally so gross as to impair normal functioning, but are observable under standardized laboratory conditions. One neurobehavioral function which has been of particular interest is that of static ataxia. Static ataxia refers simply to ataxia at rest, or incoordination of balance or equilibrium. The labyrinth-vestibular sensory system is the primary mechanism for maintaining balance and awareness of one's position in space. Through a pattern of semicircular canals and other mechanical devices within the ear, the position of the head is translated from a mechanical force to a nerve impulse within the ganglion cells of the internal auditory canal and reaches the brain stem via the vestibular nerve, which in turn connects, by long fiber tracts, to the cerebellum and oculomotor nuclei. (The cerebellum is a portion of the brain connected to the brain stem that does not initiate movements, but works closely with the motor cortex and other areas to coordinate balance and posture.) Impulses from the retina are coordinated by the oculomotor mechanism to supply information about movement and position of the body relative to the environment. Impulses from proprioceptors of joints and muscles are also essential to all reflex, posture, and volitional movements. Thus, the cerebellum and certain centers in the brain stem coordinate sensory data and provide for postural adjustment used in standing steady or in locomotion.

A report by Lipscomb, Carpenter, and Nathan (1979) that measures of static ataxia differentiated young men with or without a family history of alcoholism was based on somewhat of a serendipitous finding. The design of that study included administering alcohol to young men and determining their tolerance to ethanol using a variety of behavioral assessments both before and after the alcohol administration. The use of static ataxia measures to determine alcohol tolerance had been anticipated several years before by Goldberg (1943) in Sweden who made extensive tests of alcohol tolerance in alcoholics, heavy users, and controls. The serendipity was in finding that persons with a family history of alcoholism differed, at baseline, from those without such a history. A number of attempts to replicate this work have largely been successful. A study of 1200 school children who were administered the static ataxia test show clear differences as a result of family history (Lester and Carpenter, 1985). In our first investigation of this phenomenon, children of alcoholics, normal controls and children of depressives were found to differ on static ataxia using an apparatus patterned after Lipscomb's setup (Hegedus et al., 1984). These results were quite encouraging because they suggest a specificity of this marker for alcoholism. More recent work in our laboratory (Hill et al., 1987) included children of male alcoholics from high density families who were compared on two measures of static ataxia with children of controls confirmed the earlier reports of Lipscomb et al., Hegedus et al., and Lester and Carpenter, all of whom have found children of alcoholics to sway more than control children. These results extend the observation to include children of alcoholics who were, because of the design of the larger study, also the nieces and nephews of alcoholics. Many had grandparents and/or great aunts and uncles who were alcoholic as well. Analysis of the extended pedigrees indicated an average of 3.3 males and 0.33 female first- or second-degree relatives who were alcoholic. Thus, it would appear that the subtype of alcoholism exhibited in fathers of the high risk children assessed here is a more genetically mediated form termed "male-limited" by Cloninger. It is of interest to note that the majority of our subjects were female, suggesting that sway may be a useful psychobiological marker even among females who are selected from families in which there is a heavy loading of this more genetically mediated form of alcoholism.

Event-Related Potential Characteristics: There is an extensive literature linking chronic alcoholism to cognitive dysfunction using both neuropsychological (Goodwin and Hill, 1975; Parsons and Leber, 1982; Ryan and Butters, 1983) and neurophysiological measures (Pfefferbaum et al., 1979; Porjesz et al., 1980; Porjesz and Begleiter, 1982). A significant breakthrough in understanding the etiology of alcoholism occurred when it was established that children of alcoholics, who are at increased risk for developing alcoholism, differ from children of nonalcoholic individuals in both cognitive functioning and electrophysiological activity (Drejer et al., 1985; Gabrielli et al., 1982; Pollock et al., 1983; Begleiter et al., 1984).

Further evidence in support of a genetic contribution to neuroelectric activity, which in turn appears to differentiate those at high and low risk for developing alcoholism, comes from a variety of sources including studies examining EEG alpha activity (Gabrielli et al., 1982; Pollock et al., 1983) and event-related potentials (ERPs), particularly the P300 component of the ERP (Begleiter et al., 1984; Elmasian et al., 1982; Polich and Bloom, 1985; Steinhauer et al., 1987). P300 is a positive shift at the scalp which occurs with a latency of approximately 300 msec. If alterations in cognitive functioning and electrophysiological activity are seen in relatives of alcoholics who are not themselves alcoholic, then it is possible that some of the cognitive capacity and electrophysiological activity that distinguishes alcoholics from nonalcoholics is, in fact, antecedent to the development of alcoholism. Indicators or markers from these two domains could then be considered candidates as "vulnerability" markers rather than "episode" markers -- that is, markers for risk, as contrasted with markers for alcohol consumption per se.

P300 and Cognitive Capacity: One of the areas which appear to be a major contributor to the generation of P300 is the hippocampus (Halgren et al., 1980), a neuroanatomical site most closely associated with learning and memory functioning (Isaacson, 1975). P300 has been thought to reflect memory updating operations during information processing (Donchin et al., 1978; Donchin, 1981). Further, the latency and amplitude of P300 appears to be related to normal and impaired memory functioning (Polich et al., 1986) so that shorter latencies appear to be related to good cognitive functioning while longer latencies are related to poorer cognitive capacity. Because neuropsychological studies indicate that Family History Positive individuals do more poorly on neuropsychological tests than do Family History Negatives (Schaeffer et al., 1984), it may be the case that the P300 component reflects risk for alcoholism as it relates to more general cognitive capacity. If so, it is expected that within-family variations in cognitive capacity would be evident, with the unaffected sibling being the "outlier" of the family. If the unaffected sib were found to differ from his relatives, it would be important to determine if the variation seen was simply due to differences in alcohol consumption. We set out to answer this question by studying ERP characteristics in families where three brothers were available for testing: a pair of alcoholic brothers and one non-alcoholic brother, and their parents.

P300 Varies in Adult Siblings of Alcoholics: We find that variations in P300 are apparent in the adult siblings and parents of alcoholic individuals. While previous investigations have linked P300 to risk for developing alcoholism (Begleiter et al., 1984; Elmasian et al., 1982; Polich and Bloom, 1985; O'Connor et al., 1987), to date the most impressive demonstration of this has been in children (Begleiter et al., 1984). Prior to the present study, it had been presumed that eliciting highly significant differences between high and low risk relatives of alcoholics required that children, who had not yet become major consumers of alcohol, be included as subjects. Other studies (Elmasian et al., 1982; Polich and Bloom, 1985; O'Connor et al., 1987) employing young adults (college students) with family histories either positive (at least one first or second degree relative with reported alcoholism) or negative for alcoholism have produced less consistent results. With the initial reports (Elmasian et al., 1982; Polich and Bloom, 1985; Neville and Schmidt, 1985) that alcohol consumption altered P300, particularly the latency of the wave, it was assumed that risk could be detected most clearly in children of alcoholics who had not yet begun to drink. The present results indicate that risk status can be detected even in adult relatives of alcoholics, and that P300 may vary with risk status within families.

The findings for latency of P300 in high risk individuals reported here partly confirms and extends our previous results (Steinhauer et al., 1987). Using these tasks, we have reported that it is latency, not amplitude, that differentiates siblings of alcoholics, all of the brothers having longer latency than control subjects. With a larger sample and employing an analysis that includes the fathers and mothers of the proband alcoholic, it now appears that the unaffected sibling differs from all of his first degree relatives in having a shorter latency of P300. Differences were highly significant when the task requiring greater information processing was utilized.

The decreased latency which characterizes the unaffected sibling appears to distinguish him from his affected brothers. It might be argued that the unaffected sibling has a shorter latency than his affected brothers because the alcoholic siblings have a history of excessive drinking spanning many years which the unaffected sibling does not have. While we cannot rule out this possibility, it seems very unlikely for two reasons. First, the average age of our affected siblings was 34 years, suggesting a shorter history of drinking than is usually seen in association with alcohol-induced cognitive dysfunction. Second, correlations between alcohol use in the past week and latency of P300 were nonsignificant, suggesting that alcohol use in our affected subjects who continued to drink did not alter the latency of P300 in these subjects.

ERPs in Children of Alcoholics: Thus, although reports have been mixed where adult high risk young men have been assessed, in contrast to more positive findings for minor children (Begleiter et al., 1984; Whipple and Noble, 1986), nevertheless, we have found differences in ERP characteristics of adult men from high risk families when contrasted with low risk (control) subjects (Steinhauer et al., 1987; Hill et al., in press). The subject population studied has included individuals from high density families where multiple cases of alcoholism are found. This is in contrast to the usual methodology employed in the family history positive/family history negative strategy where a single first degree relative qualifies one for membership in the "high risk" (family history positive) group. Realizing that residual effects of alcohol consumption might have subtle effects on information processing, though correlations between P300 amplitude and latency and recency of alcohol use have not been found (Hill et al., in press), to rule out any confounding effect of alcohol use, we have, in this study, begun testing the minor children of subjects previously described (Hill et al., in press) who represent the third generation for whom ERP data is being collected.

Two groups of children were tested: children of alcoholic fathers and children of normal controls. The ,coholic fathers were selected from treatment facilities if they had a diagnosis of alcoholism and were free of other DSM-III (Axis I) psychopathology. Families were included only if all first degree relatives met criteria for absence of other psychopathology with the exception of sociopathy which was present in some families.

Evaluation of event-related potentials in children of alcoholics compared to children of controls revealed two major findings: an enhancement of frontal negativity at N250 in the children of alcoholics and deviations in P300 amplitude and latency under conditions of minimal task engagement among the high risk children. The N250 refers to a negative wave that occurs at approximately 250 milliseconds after the onset of a stimulus that is to be processed. Increased frontal negativity in high risk children compared to controls had not been reported previously. The decrease in P300 amplitude seen in our more difficult discrimination tasks confirmed reports of Begleiter et al. (1984) and others.

A frontal maximum N250 for children as contrasted with a vertex maximum in adults has previously been reported by Friedman et al. (1984). Friedman et al. noted the prominence of the frontal N250 in children, which decreased in negativity not only through adolescence, but which they also observed in other data (e.g., Pfefferbaum et al., 1980) to continue to decrease in negativity throughout the life span. This frontal negativity has been viewed as being associated with the development of more posterior (parietal) Slow Wave, and its decrease has been suggested as reflecting maturational development (Friedman et al., 1984). Consequently, the findings for greater negativity in the children of alcoholics in the present study suggest an interesting hypothesis: the high risk children may be showing an electrocortical sign of maturational lag. While the groups did not differ in age, it would be most critical to examine larger groups of high risk children and controls at each of several ages. Larger samples are also needed to control for possible sex differences. Friedman et al. (1984) noted greater negativity for N250 among males than females, although in the present study almost equal numbers of males and females comprise each group.

Blood Markers: The most convincing evidence that alcoholism is transmitted genetically, other than demonstrating a specific biomedical gene product, would be to demonstrate genetic linkage of an alcoholism susceptibility (AS) gene(s) to a well-defined polymorphic marker. Polymorphic markers are those that commonly exist in the population in two or more discrete forms. For a potential marker to be most highly informative, it should be reliably detectable as well as independent of the disease state, that is, it is not influenced by whether or not the at risk individual is currently affected by the illness. Ideally, it should be localized to a particular chromosomal region.

A number of polymorphisms in man have been found in blood (Race and Sanger, 1975) making it possible to attempt to find genetic linkage or association between alcoholism and these markers. Most studies have concentrated on either the ABO or ABH secretor systems (Swinson and Madden, 1973; Hill et al., 1975; Winokur et al., 1976), though two have considered other markers (Hill et al., 1975; Kojic et al., 1977) located on various chromosomes. Association involves showing that the frequency of a particular allele differs in a population of affected individuals from that seen in a normal control population. Linkage assesses the co-segregation of a particular disorder within families with a marker. If linkage is demonstrated, it is assumed that the marker -- in this case, a particular blood group -- is on the same chromosome as the AS gene and is in sufficient proximity to the marker so that the loci fail to follow Mendel's law of random assortment. (It is possible to have genetic linkage between an AS gene and a marker without there being association between those loci.)

We investigated the possibility of genetic linkage between the AS gene and six blood group markers: Rhesus and Duffy on chromosome 1, MNS on chromosome 4, ABO on chromosome 9, Kidd on chromosome 18 and Kell (unassigned), among 30 nuclear families chosen because there was a pair of alcoholic brothers present. Only those families with a demonstrated absence of other psychiatric disorders in first and second degree relatives (e.g., affective disorders, schizophrenia) were included. Personal psychiatric interviews were conducted for all family members who participated in blood typing (N = 124) and who were first-degree relatives, allowing for determination of whether or not the subject met accepted criteria for alcoholism. All probands and affected sibs included in the present analysis met these criteria. Family history information was sought for second- and third-degree relatives of the proband and those first-degree relatives who were deceased, or did not participate in the face-to-face interview and blood draw. Presence of alcoholism in these individuals was diagnosed only if at least two family members reported problems associated with drinking. In this way, pedigrees were extended to include a total of 220 first-degree, 348 second-degree and 288 third-degree relatives. This information was used to complete a segregation analysis to determine a suitable genetic model to use for the linkage analysis.

A suggestion of genetic linkage between the MNS blood group and a putative alcoholism susceptibility gene was found using two methods to evaluate the genetic linkage, sib-pair and LIPED analyses. It is possible that even higher lod scores might have resulted if a more homogeneous sample of families could have been studied.

Further study is required to firmly establish both the genetic model for the alcoholism susceptibility gene and genetic linkage between this gene and the MNS blood group. As part of a larger study, a number of high density pedigrees are being ascertained. Therefore, we intend to collect more families to determine if this finding can be replicated.

SUMMARY AND CONCLUSIONS: THE VULNERABILITY MODEL

As is abundantly clear from the foregoing review, each model makes a unique contribution to understanding the factors that may be responsible for the development of alcoholism. Each model fails, however, to explain why one individual becomes an alcoholic and another does not. Even if alcoholism were completely determined by genetic factors, we could expect that some individuals would carry the genotype but not express the disorder because of incomplete penetrance. Clearly, there is a need to merge the various theories and models into a supermodel to adequately explain the etiology of alcoholism. We propose that a vulnerability model may offer greater explanatory power when considering the diverse expression of alcohol abuse patterns and outcome. Rather than postulating that alcoholism is a disease or at least a biological condition, on the one hand, or a defect in social learning on the other, the vulnerability model attempts to incorporate relevant findings from both the biological and psychosocial domains.

Though this view may seem heretical to those accustomed to viewing alcoholism as a chronic disease state, in which the alcoholic is considered to be, lifelong, a "recovering alcoholic," we propose that alcoholism is an episodic disorder, developing in the vulnerable individual when subjected to exogenous or endogenous stressors. However, a minority of individuals who are biologically vulnerable and who undergo stressors develop episodes. In fact, some highly vulnerable individuals never develop an episode of excessive drinking at all.

Among the moderating variables which may prevent development of an episode are: the individual's personality and cognitive capacity, a supportive (perhaps nondrinking) social network, and a favorable ecological niche. That not all vulnerable individuals develop episodes is illustrated in the Danish adoption study. The majority (73%) of offspring of alcoholics adopted away shortly after birth (Goodwin et al., 1973) did not develop alcoholism as adults, though of course the risk was much higher among this group than in a matched control group (18% versus 5%) by age 30 when they were assessed.

The importance of identifying biological markers for risk is readily apparent. With these tools we may one day be able to describe quantitatively the lifetime risk for developing alcoholism for particular individuals. With this information in hand, the individual can make a much more informed decision about whether or not he or she wishes to drink at all, and if so, to be aware of the need for moderation and monitoring of his or her drinking behavior.

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Written Testimony of

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Hearing on Alcoholism (Etiology)

INTRODUCTION (including some definitions of terms)

Two-thirds of the population in this country drink alcoholic beverages, but there is wide variation in the quantity and frequency of drinking. It is estimated that 10% of drinkers (6.5% of the total population) accounts for 50% of all the alcohol consumed; they also experience the majority of the adverse psychosocial and medical consequences. Thus alcohol abuse and alcoholism, both pathological patterns of alcohol consumption, are disorders of a dimensional rather than a categorical nature. The diagnostic entities of alcoholism and alcohol abuse refer primarily to the central nervous system effects of alcohol, as manifested by the behavior of repetitive and excessive use of alcohol, the neurological and behavioral consequences of acute intoxication, and the neuroadaptive changes associated with chronic alcohol consumption.

Alcoholism can be defined as a disorder characterized by persistent and progressive patterns of abnormal alcohol-seeking behavior that, over time, result in tolerance and in psychological and physiological dependence. This definition embodies the construct of the "alcohol dependence syndrome" proposed by Edwards and Gross in 1976 and is consistent with the operant approaches accepted in the study of other addictive drugs. The alcohol dependence syndrome has as its cardinal feature impaired control over alcohol use and assumes that tolerance and dependence play important psychophysiological roles. The development of alcoholism and its course are influenced both by genetic predisposition and environmental factors. The consequences of alcoholism include a variety of physical and mental disabilities as well as impairment in social functioning.

It is important to distinguish between primary and secondary alcoholism. The latter is heavy alcohol use manifesting elements of the alcohol dependence syndrome that occurs during the course of another psychiatric disorder, e.g., depression, schizophrenia or bipolar affective disorder. Alcoholism in this setting might be symptomatic of the primary diagnosis and the course is likely to be that of the primary disorder, not alcoholism. Seventy to eighty percent of all alcoholics exhibit primary alcoholism.

Alcohol abuse can be defined as a heterogeneous set of behaviors characterized by repetitive patterns of heavy drinking which, while not yet producing dependence, lead to impairment in psychological and/or social functioning and/or health because of intoxication. These patterns of drinking need not be progressive, and may remit with time. Their occurrence and course are strongly influenced by sociocultural and psychological factors and by stages in psychosocial development. As example, alcohol abuse in adolescents can lead to negative consequences, such as poor school performance, family problems and arrests, but this behavior and the associated complications usually ameliorate with age and with changes in life events and environment.

Alcohol abuse and alcoholism are among the nation's most serious health problems. It has been estimated that 10.6 million people over the age of 18 are alcoholic and 7.3 million are alcohol abusers. Some 4.6 million adolescents also experience negative consequences of alcohol use. Children of alcoholics, a high risk group, are estimated to number 28 million in this country alone. Studies have shown that alcohol abuse and alcoholism affect between 11 and 16% of the population at some time in their lives. They affect 19-29% of males and 4-6% of females. Alcoholism is seen in all socioeconomic and ethnic groups. The homeless or skid-row alcoholic actually represents less than 7% of all alcoholics in this country.

Since not all who drink alcoholic beverages become alcoholic and experience alcohol-related disabilities, the determinants of susceptibility must reside in host and environmental factors. As has been discussed by others who are testifying today, there is now increasingly convincing evidence in support of a biobehavioral conceptual model of the etiology of alcoholism. In this model, the disorder is perceived as arising from the combined effects of genetic/biological vulnerability and psychosocial risk. Drinking, or alcohol-seeking, is a metric trait and alcoholism lies at the extreme, high-end of this quantitative measure. Metric traits almost always have multiple genetic determinants; thus the extent of genetic loading of biologic risk for alcoholism differs in different individuals. Added to this kind of variability is the wide range of options for exposure to the psychosocial risk factors of heavy drinking provided by modern society. Further, environmental risks change when stages of psychosocial development and life-events change. It is not surprising, therefore, from the combination of the kinds of genetic and environmental variability described above, that there is a wide array of patterns of expression of the disorder alcoholism. In the search for understanding of underlying mechanisms and for rational basis of potential therapy, it is important to focus our attention upon the final common pathway for expression of this disorder, alcohol-seeking behavior.

In this context, it is relevant to examine what kinds of psychosocial and biological variables can affect drinking behavior. Environmental factors, e.g., certain kinds of sociocultural norms, family/peer influences, and stages of psychosocial development, are known to foster the initiation of drinking as well as its continuation and progression. The development of alcohol-related problems and changes in life events can encourage moderation or cessation of drinking. Although no obvious biological factors other than personality differences are known to influence initiation, a number of biological responses to alcohol as a drug, such as the reinforcing, neuropsychopharmacological effects of alcohol, the development of tolerance, and physical dependence, can promote continuation and progression. On the other hand, physiologically aversive effects of ethanol and medical complications may deter drinking or encourage moderation. In this context, a brief discussion of these effects of ethanol is appropriate.

Ethanol is primarily a central nervous system depressant and the degree of depression produced is directly related to the amount consumed. However, when blood alcohol concentrations (BACs) are low to moderate (less than 50 mg%), behavioral stimulation and a sense of well-being or euphoria may occur. This state is ascribed to the depression of the activity of certain brain structures which ordinarily are inhibitory, and is generally considered to be the rewarding or reinforcing feature of social or moderate drinking. As BACs rise above 50 mg%, measurable impairment in cognitive and psychomotor functioning appears, followed by severe motor incoordination, drowsiness and unconsciousness. Death can occur because of respiratory depression with BACs as low as 300 mg%. It is notable that there is a great deal of between-individual variation in all of the above responses to alcohol. Studies in animal models indicate that this variation is determined to a large degree by genetic factors (vide infra). However, it should be emphasized that an individual's response to ethanol is also influenced by environmental factors and by expectancy effects. The social context of drinking and expectancy can heighten or lessen both the stimulatory and depressant effects of ethanol.

Tolerance describes the ability of an organism to adapt to the effects of a drug. One way is to increase the rate of elimination of the drug. The rate of ethanol elimination can increase as much as 30% with chronic exposure. This is pharmacokinetic or metabolic tolerance. The neuroadaptive response to acute and chronic ethanol exposure is called pharmacodynamic or neuronal tolerance. Neuronal tolerance is evidenced behaviorally by an improvement in performance in a test conducted at the same blood or tissue level of ethanol. Tolerance is expressed at the cellular level by changes in the composition of cellular membranes, in properties of membrane-bound enzymes, and in the metabolism and activity of various neurotransmitter systems. Tolerance can also occur because of learning, developed in the context of environmental cues associated with the administration of ethanol. This is referred to as environment-dependent, conditioned or learned tolerance. Individuals differ widely in their capacity to acquire tolerance.

Physical dependence refers to the compensated state of cellular and metabolic functioning that develops when an organism is chronically exposed to elevated drug levels in the body. It has been hypothesized that a decrease in neuronal membrane fluidity among other metabolic or enzymatic changes may underlie this adaptive response, because ethanol increases membrane fluidity. When ethanol consumption decreases significantly or ceases, the compensated nervous system becomes hyperactive in the absence of ethanol, giving rise to the abstinence or withdrawal syndrome, which is characterized by behavioral excitation, tremulousness, delirium, hallucinations, and seizure activity.

HERITABILITY OF RESPONSES TO ETHANOL AND EFFECT ON DRINKING BEHAVIOR

In looking at the biological responses to ethanol that can serve as feedback loops to influence drinking behavior, it is noteworthy that a number of them, as well as drinking behavior itself, exhibit a large degree of between-individual variability which is, in part, genetically determined. This has been shown in humans from studies comparing identical and fraternal twins and comparing different racial groups. These include: frequency of drinking, consistency of drinking behavior over time and change, the response of the brain to ethanol as revealed by eeg measurements, systemic reactivity to ethanol as evidenced by the alcohol-flush reaction (vide infra), alcohol metabolic rate, and perhaps also the incidence of alcohol-induced liver disease and brain damage.

Recent studies in humans have shown that genetic differences in alcohol elimination can have a profound influence on drinking behavior and the development of alcoholism. The major avenue of alcohol elimination from the body is metabolic conversion, first to acetaldehyde, then to acetate, and finally to carbon dioxide and water. The enzymes that catalyze the first two reactions are alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH), respectively. There are genetic variant forms of these enzymes and they differ dramatically in catalytic properties. For example, humans have more than five different forms of ADH and the genetic variants of one of these forms (β-ADH), seen in Orientals and American Blacks, are more that 20- and 7- times more active, respectively, than the corresponding form seen in White populations. These differences as well as polymorphism in the other alcohol dehydrogenase forms (e.g., γ-ADH), are thought to underlie much of the three-fold variation in alcohol elimination rate observed among humans.

There is also genetic polymorphism involving ALDH and a genetic variant of one of the ALDH forms has been shown to cause alcohol intolerance. This is the so-called low-Km mitochondrial form of ALDH which is the principal enzyme form that metabolizes acetaldehyde. Acetaldehyde is a very toxic substance and the low-Km (high affinity) mitochondrial ALDH in liver is responsible for keeping acetaldehyde to a very low level (>10 μM) in the body. The genetic variant seen in about 50% of Orientals is an inactive enzyme - a null mutation. The lack of activity of this enzyme leads to elevated acetaldehyde levels after ethanol consumption which in turn leads to a toxic reaction. The syndrome consists of facial and upper extremity flushing, rapid heart rate, vasodilation, headache, nausea, vomiting, and, in severe cases, hives, edema and hypotension. The alcohol-flush reaction is identical to the alcohol-Antabuse reaction. Antabuse (disulfiram) is a drug used to treat alcoholism. This drug inactivates ALDH. As result, if the alcoholic patient taking Antabuse drinks, he/she develops the unpleasant alcohol-Antabuse reaction. These reactions are thought to be mediated by the release of histamine, prostaglandins and vasoactive peptides from cells such as mast cells, provoked by the high circulating acetaldehyde levels.

The alcohol-flush reaction is protective against heavy drinking. Thus inheritance of this enzyme defect in acetaldehyde metabolism is a negative risk factor for developing alcoholism. Several studies from Japan have now shown this to be the case. Whereas the alcohol-flush reaction and the inactive variant form of mitochondrial ALDH is seen in 50% of the general population in Japan, they are seen in only 5-10% of Japanese alcoholics, with or without liver disease. Population surveys have also shown that those who flush tend to drink less per session and less frequently than those who do not. There is, however, a great deal of between-individual variation in severity of the alcohol flush reaction. This may be the reason that some people who have the alcohol-flush reaction still drink. We believe that the variation in severity of the flush reaction is dependent upon whether one is homozygous or heterozygous for the variant allele of ALDH and upon the genetic polymorphism of ADH, the enzyme that generates acetaldehyde. In the past, determination of the enzyme phenotype and supposed genotype has depended upon obtaining liver specimens. In the last six months, investigators at the Indiana Alcohol Research Center, which is funded by NIAAA, has succeeded in directly genotyping both the ADH and the ALDH by using white blood cell DNA. Thus we are no longer dependent upon using liver specimens for determining the phenotype and deduced genotype of the enzymes of alcohol metabolism a given individual possesses. The above hypothesis, therefore, can now be directly tested by using a blood specimen. We will now also be able to test directly the hypothesis that genetic polymorphism of ADH and ALDH are linked to some of the medical complications of chronic ethanol consumption, such as cirrhosis, pancreatitis, dementia.

Humans are also known to differ in the degree to which they are behaviorally impaired by the same dose of alcohol and in the degree to which they develop tolerance. Although definitive studies of the heritability of these traits in humans have not yet been performed (they are clearly difficult to perform for ethical reasons), studies in experimental animals have shown an interesting link between the development of neuronal tolerance and alcohol-drinking behavior.

As with humans, drinking behavior, the innate sensitivity of brain to the depressant actions of ethanol, and alcohol metabolic rate are significantly influenced by genetic factors in experimental animals. Interestingly, tolerance development both in terms of how much and how long they last, and the ease with which physical dependence develops and its severity, have also been shown to be genetically determined in experimental animals.

As with humans, animals also exhibit a large degree of variability in their response to ethanol that are, in part, genetically determined. This has been shown through studies in inbred strains of mice and in selectively bred lines of mice and rats. The finding of a difference in a trait between inbred strains is prima facie evidence for a genetic determinant of that trait. Experimental animals can also be genetically manipulated by selective breeding. This process can systematically segregate the genes for a chosen trait into or out of lines of animals so that animals with high and low measures of that trait are developed. This clearly is another way to substantiating the heritability of a trait, and the contrasting high and low lines can then be used to study the biochemical and physiological mechanisms underlying that trait.

Our research laboratories at the Indiana University School of Medicine have been working the past 12 years on developing animal models for the study of alcoholism. It has been our contention that selective breeding for the trait of high alcohol drinking preference can yield an animal model of alcoholism, because the final common path in expression of the disorder is aberrant alcohol-seeking behavior, regardless of etiology. It is well known that most experimental animals avoid drinking ethanol solutions that are 10% (v/v) or higher in concentration, when food and water are concurrently available. However, some strains of outbred laboratory rats do show a wide range of individual variation in alcohol preference, and lines of rats exhibiting high and low alcohol preference have been raised through bidirectional selection in out laboratories and in Helsinki, Finland. The line of alcohol-preferring rats developed in Indiana, the P line, has now been systematically characterized with respect to its drinking behavior. The data indicate that it satisfies all the perceived requirements for an animal model of alcoholism. Comparison of the effects of ethanol on P rats and on NP rats, a line simultaneously selected for alcohol nonpreference (aversion), has revealed differences that have provided interesting insights into the biological basis of aberrant alcohol-seeking behavior.

The suggested essential requirements of an animal model of alcoholism are:

1. Ethanol should be self-administered orally, in preference to water or other solutions having the same caloric value and palatability.

2. The amount consumed should produce measurable blood alcohol concentrations (BACs).
3. Ethanol should be positively reinforcing, i.e., the animals should be willing to overcome obstacles or work to obtain the alcohol.
4. The rewarding effect of ethanol should be accountable in large part, if not entirely, by the CNS pharmacological actions of ethanol or its metabolic products and not because of its caloric value, taste, or smell.
5. Consumption should ultimately lead to
    a) repeated episodes of intoxication
    b) tolerance
    c) physical dependence

We have shown that the P rats satisfy all of the above criteria. Further, we have shown that the P rats will work (through bar-pressing) in order to obtain the alcohol even at concentrations as high as 30%. This occurs even when food and water are freely available. Thus alcohol serves as a reward or a reinforcer to the P rats. By contrast, the NP rats would not bar-press for alcohol at all at concentrations higher than 5%. Further, it is the post-ingestive, pharmacological actions of ethanol that are reinforcing since the P rats, but not the NP rats, will self-administer alcoholic solutions as high as 40% directly into the stomach. The reason for the difference between the lines is that, whereas the P rats find low to moderate doses of ethanol to be rewarding, the NP rats are less affected by the reinforcing actions of ethanol. Instead, the NP rats begin to develop aversion to ethanol at a much lower dose than the P rats. Importantly, the P rats also develop tolerance to the high dose aversive effects of ethanol more rapidly and remain tolerant for a longer period of time than do the NP rats.

These differences in reaction to ethanol are clearly relevant to our understanding of how alcohol abuse develops. We hypothesize that individuals with a genetic propensity for alcohol abuse and alcoholism find alcohol in low to moderate doses to be rewarding behaviorally and that they develop tolerance to the high-dose aversive effects of alcohol more effectively. This hypothesis should be directly testable in humans. For example, are children of alcoholics as a group more likely to exhibit these responses than children of nonalcoholics? The ongoing studies from Dr. Schuckit's laboratory in San Diego would suggest that they are, but more in depth studies are needed.

Our studies in P and NP rats have also shown that alcohol preference is associated with a lower content of the neurotransmitter serotonin in certain brain regions, especially those in the so-called brain reward circuitry. Interestingly, the administration of a drug that blocks serotonin metabolism (fluoxetine, a serotonin reuptake inhibitor) curbs the appetite of the P rats for alcohol. Other drugs such as desipramine and naloxone also can suppress the alcohol consumption of the P rats. Thus the involvement of other neurotransmitter/modulator systems is likely. These include the dopamine and enkephalin systems as well as GABA. Clearly the animal model can be useful for the screening of potential drugs than might reduce alcohol craving.

IMPLICATIONS FOR TREATMENT AND PREVENTION

The recent basic and clinical research on the alcohol-flush reaction has clearly shown that there are negative risk factors for heavy drinking and alcoholism that are genetic and metabolic in nature. The inheritance of the gene for the inactive variant of the low-Km mitochondrial ALDH is a classic example of a pharmacogenetic disorder. It happens that pharmacotherapy based on this principle was developed empirically before the biochemistry and genetics of the alcohol-flush reaction were elucidated. We now know that Antabuse, Temposil and other ALDH inhibitors have a place in the treatment of alcoholism, but their efficacy is limited by problems in compliance. It would appear that drugs that would alleviate craving or alcohol/drugseeking behavior might be the direction for the future. The studies in animal models, and limited studies in humans, suggest that agents which act to raise brain serotonin and dopamine, to block opioid receptors and to stimulate GABA-benzodiazepine receptors might have promise.

The studies in animal models and in children of alcoholics have suggested that genetically susceptible individuals do not respond the same way as the average person. They may, as a group, be less sensitive to the aversive effects of ethanol and be more able to develop tolerance than those without a genetic predisposition. If this can be established definitively, then an important prevention strategy can be developed. The affected individuals should be identified and primary and secondary prevention efforts should be directed at the identified susceptible individuals to adopt moderate drinking practices or abstinence.

The critical issue here is identification. Although family history is currently still the best predictor of future alcohol abuse and alcoholism, not all children of alcoholics inherit the genetic susceptibility. Therefore, it should be a high research priority to develop tests that would differentiate those who are genetically susceptible. In this regard, a number of promising phenotypic markers and genetic markers for the enzymes of alcohol metabolism have been discovered. The field is therefore, at the stage at which family studies to identify genetic linkage and relevant genetic/phenotypic markers should begin to be conducted. This kind of large research program requires a collaborative effort on the part of several research centers in the country and requires long-term funding. Support for research from federal agencies should consider this direction as a priority item.

The identification of Type II alcoholics should be a high priority item because it is these individuals who tend to be polydrug abusers and have social complications of alcohol use. They are also the ones, once identified and treated, who do very well subsequently in society. Accordingly the kinds of research proposed above are cost-effective.

Predictions of Elderly Drug Abuse[†]

MEYER GLANTZ, PH.D.[*]

Drug abuse is typically associated with adolescents and young adults. With the "aging of the population," more public attention is being directed to the elderly and the possibility of drug abuse by the elderly has become a matter of concern. The elderly, those adults who are 65 years or older, are commonly assumed not to be users of illicit drugs and, while they are generally recognized as having high levels of usage of legal drugs, they are usually assumed to use those drugs in a licit, prescribed manner. Although there have been reports that some physicians overprescribe psychoactive drugs for the elderly, that some residential care facilities for the aged make frequent errors in administering medications and that some use "chemical restraints," as well as reports that the elderly are at high risk for drug interactions due to the number of different medications typically prescribed for older adults (e.g., Petersen, Whittington & Beer 1979), these are problems of drug misuse and not drug abuse. There have also been reports that some elderly adults do not use licit medications in the manner prescribed by their physicians or indicated in the use instructions for over-the-counter medications; these instances of noncompliance are also typically assumed to be attributable to misuse. In fact, some individuals feel that the term "drug abuse" cannot be properly applied to the drug usage patterns of the elderly and that only the term "drug misuse" can be accurately applied. This presumes that any inappropriate usage of drugs by the elderly is usually inadvertent, that the elderly do not typically intend to use drugs in an inappropriate fashion.

To avoid any confusion, the terms "drug misuse" and "drug abuse" will be defined according to the definitions adopted by the federal Strategy Council on Drug Abuse (1979):

> *Drug abuse* is the non-therapeutic use of any psychoactive substance, including alcohol, in such a manner as to adversely affect some aspect of the user's life. The substance may be obtained from any number of sources – by prescription, from a friend, over-the-counter, or through the illicit market. The use pattern may be occasional or habitual.
>
> *Drug misuse* is the inappropriate use of drugs intended for therapeutic purposes. This includes inappropriate prescribing or use of drugs resulting from: (a) lack of knowledge on the part of the physician; (b) errors in judgment by the physician, including drugs prescribed when there is a preferable or safer alternative treatment (such alternatives may include non-drug treatment); (c) use by a patient of a prescription drug not under the supervision of a physician or not in accordance with the instructions of the physician or the information provided with the drug; and (d) self-medication

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[†] This paper does not necessarily represent the views of the National Institute on Drug Abuse.

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by a patient with a drug (over-the-counter or prescription) inconsistent with the label information.

It is not surprising that there is a general reluctance to consider the issue of elderly drug abuse; the implications of even the possibility of such a problem are extremely unpleasant. Nevertheless, there are indications that the elderly are susceptible to substance abuse and that they will be increasingly at risk for at least the next decade. Research in this area is really just beginning and the relevant literature is limited, often inconclusive and sometimes contradictory. Despite this, it may be possible to hypothesize a model of elderly drug abuse by supplementing the available literature on elderly drug misuse and abuse with inferences from the literature on elderly alcoholism and the literature on adolescent drug abuse. As the first step in the attempt to develop a model of elderly drug abuse, some of the literature on the use of licit and illicit drugs by the elderly will be considered. This literature has been reviewed more fully by Petersen, Whittington and Beer (1979).

ILLICIT DRUG USE

One form of drug abuse is characterized by the abuse of illicit drugs. It is generally assumed that the elderly do not typically use illicit drugs, and this seems to be borne out in research (Cisin, Miller & Harrell 1978). This suggests that drug abuse, which occurs among the general elderly population, is likely to involve legal substances which are being used in an abusive fashion. There is, however, a small group of elderly opiate addicts. The number of publicly visible elderly opiate addicts is smaller than would be expected by projections from the number of addicts who were known to be using opiates in the past and who would have now aged into their elderly years. This type of observation led Winick (1962) and others to postulate a "maturing out process" in which addiction ceases spontaneously due either to the duration of the addiction or to the age of the addict. Winick felt that most people have fewer stresses as they approach middle age and, therefore, they are less likely to rely on drugs as a coping mechanism. Maddux and Desmond (1980) found that only 22 percent of the 248 addicts in their 16-year longitudinal study "matured out" of their addiction. Capel and his associates (Capel & Peppers 1978, Capel et al. 1972) found that, while some addicts do mature out, the majority adapt and conceal their habits by using other drugs, such as Dilaudid®, by decreasing their daily level of drug usage and by occasionally substituting more legally available substances such as alcohol or barbiturates. This has several implications. First, it implies that illicit drug abuse by the elderly and perhaps all elderly drug abuse, follows a pattern which, compared to the drug pattern of younger abusers, involves less stress and less risk of criminal apprehension, social disruption or physical danger and which is less detectable. Second, it implies that when a reliance on drugs is developed, the dependence may persist even if the drug used originally is not obtained. Third, it implies that the population of elderly illict drug users will increase proportionately over the next several decades. The percentage of pre-elderly opiate users is disproportionately high and, as those users reach old age, the proportionate number of elderly illicit drug users should increase. Capel and Peppers (1978) and Pascarelli and Fischer (1974) have already reported an aging of the addict population and they predict a sharp increase in the number and percentage of elderly addicts in the next decade. There has also been a report by Singh and Knezek (1978) that, contrary to previous findings, the 1976 Client Oriented Data Acquisition Process (CODAP) client sample became opiate addicts in their late 30s and early 40s which is considerably later in life than was found by previous CODAP studies. This upward shift may increase the likelihood that the career of current opiate addicts will actively extend into that group's old age and it suggests that acute patterns of abuse may begin later in life than is often assumed.

LICIT DRUG USE AND MISUSE

Surveys of the epidemiology of legal drug use reveal that the elderly use very large amounts of prescription and over-the-counter drugs (e.g., Petersen, Whittington & Beer 1979). The elderly have high levels of use of psychotherapeutic drugs, such as tranquilizers and sedatives, and they also maintain high levels of use of drugs with associated psychoactive effects, such as non-narcotic analgesics and antihistamines (e.g., Warheit, Arey & Swanson 1976). Such high levels of drug use do not necessarily imply either drug misuse or drug abuse. It may well be that a high level of medication use by the elderly is appropriate, since the elderly are considerably more likely than any other group to have serious chronic diseases and disabilities. Certainly this is true, but it leaves the question of whether it is appropriate for the elderly to be taking such high levels of the subset of drugs which are psychoactive. It might be argued that such a high level of psychoactive drug use is appropriate, as the elderly also demonstrate a very high incidence of psychiatric problems. Although this is the case (e.g., Pfeiffer 1978), and regardless of whether or not physicians and care providers are believed to be overprescribing and over-relying on these medications, high levels of drug use do have implications for drug abuse. If the elderly are in such distress that they require such high levels of medication, then they may also be in sufficient distress to engage in self-medication in an abusive manner. The use of prescription drugs by the elderly has been shown to be associated with coping problems. Gutmann (1977), for example, found that the use of legal drugs was consistently related to lowered self-esteem and low self-assessment of personal capabilities. It is possible that the prescription of such high levels of psychoactive drugs may: (1) encourage the elderly to think of, and come to rely on, drugs as problem-solving mechanisms; (2) seem, at least to some elderly people, that family, physicians and other care providers sanction the use of drugs as coping mechanisms; and (3) substitute for the development of alternative coping mechanisms and attempts to seek tangible solutions to problems. If drug use is a major coping mechanism for the elderly, it may provide a foundation for drug abuse, and the high levels of prescribed drugs may provide a readily available source of drugs which could be abused by the hoarding, sharing and taking of the drugs with alcohol and/or over-the-counter medications and by the obtaining of multiple prescriptions for the drugs. If overprescription is taking place, then some elderly people who might otherwise become drug abusers might be able to obtain an abusive level of drugs by simply cooperating with the misuse. Serious drug misuse involving over-medication has already been clearly demonsrrated in a large number of nursing homes and geriatric facilities (e.g., U.S. Senate Subcommittee on Long-Term Care 1975), and the problem may extend to the noninstitutionalized elderly.

There is considerable controversy over whether or not the elderly practice drug misuse. There are some studies which have found that the elderly comply with prescription directions (e.g., Stephens, Haney & Underwood 1980) and some studies which have found that the elderly are much less likely than other age groups to follow prescription instructions (e.g., Brand, Smith & Brand 1977). Although these noncompliance studies have usually not included a representative sample of the elderly and a number have suffered from problematical assessment techniques (for example, assessing compliance by simply asking the respondents if they comply with prescription instructions), these studies have typically found that noncompliance is fairly common and that the most common form of noncompliance by the noninstitutionalized elderly is the underuse or omission of prescribed medication. Unfortunately, there is also evidence of misuse in forms which have the potential to involve abuse. Taking drugs in combination with alcohol is apparently a frequent occurrence. Guttman (1977), for example, found that approximately half of the respondents in his study reported using some type or combination of legal drugs and over-the-counter drugs in combination with alcohol. The elderly also take large amounts of over-the-counter drugs in addition to their prescribed medicines and it seems that they often do so without consulting their physician or pharmacist (James 1979). Some over-the-counter drugs have psychoactive effects when taken in combination with other drugs or alcohol or when taken at very high doses. For some drugs, particularly those which are psychoactive, there is some evidence that, compared with younger adults, the elderly may experience an increased level and duration of effect (Friedel 1978; Greenblatt et al. 1978; Klotz et al. 1975; Salzman, van der Kolk & Shader 1975). In addition, the elderly are likely to be more sensitive to alcohol, experiencing an increased intensity and period of effect (Vestal et al. 1977; Wood 1976; Sun & Samorajski 1975; Wiberg, Trenholm & Caldwell 1970). This heightened sensitivity to some drugs and to alcohol may increase the possibility of over-medication, drug interactions and side effects. There are reports that some of the elderly use prescribed drugs in erratic fashions, that they hoard drugs past the intended period and that they share drugs with other elderly people (James 1979; Green 1978; Doyle 1976). There are some reports which, though based on unrepresentative samples, suggest that some elderly individuals simultaneously acquire multiple prescriptions for psychoactive drugs by using multiple physicians and/or pharmacies (e.g., Pascarelli & Fischer 1974). While some instances of these forms of misuse may involve inadvertant inappropriate use, it is likely that in some, if not many cases, abuse is involved. Further, as abusive use of alcohol and some drugs may take place at dose levels and schedules of use which would not be considered abusive use for younger adults, both care providers and researchers, whose criteria for the determination of drug abuse may be based on younger abusers, may fail to detect some elderly substance abuse and may underestimate its extent.

There have been no studies which have directly investigated the abuse of licit drugs by the elderly. There have been a few studies of elderly subjects which investigated licit drug use and which might have detected some forms of abuse (e.g., Guttmann 1977) and some studies which investigated certain forms of elderly drug abuse using indirect or limited assessments (e.g., Stephens, Haney & Underwood 1980; James 1979). These studies have not reported findings of extensive drug abuse among the elderly. However, inferences from these studies about the extent of elderly drug abuse must be qualified by the limitations of the research. These studies usually have not included representative samples of the elderly and they may be biased in ways which underrepresent drug abusers. Perhaps more of a problem is that these studies have used research tools which do not assess the full range of the potential patterns of elderly drug abuse; the patterns which are considered have often been assessed using measurements of questionable validity which, as an additional problem, do not typically attempt to distinguish between misuse and abuse and which categorize all inappropriate or medically contraindicated use as misuse. There have been a number of studies which have attempted an indirect measure of elderly drug abuse. Following the assumption that misuse and abuse often lead to medical emergencies, some researchers (e.g., Inciardi et al. 1978; Heller & Wynne 1975) have examined drug-related emergencies reported in hospitals and emergency rooms. These studies have consistently found that the elderly are underrepresented in the population of those with identified acute drug reactions. However, these studies might underestimate the total number of elderly who experience drug-related emergencies as some older people might be unwilling or unable to seek help in a clinical setting (e.g., Petersen & Thomas 1975). While the studies assessing drug abuse among the elderly might underestimate the extent of elderly drug abuse, it is likely that the elderly, compared to younger age groups, are indeed less likely to abuse drugs. This does not imply that elderly drug abuse does not exist as a serious problem involving a large number of elderly adults.

In summary, the literature on elderly drug use indicates that the elderly are at high risk for drug misuse and that they may also be at considerable risk for drug abuse involving legal drugs. The abusive use of drugs by the elderly may be associated with coping problems. While the literature on elderly drug abuse does not address many important questions, it may be possible to develop a more complete model of elderly drug abuse by building on hypotheses derived from the literature of a related problem, elderly alcohol abuse.

ALCOHOL ABUSE

Elderly alcohol abuse is a more widely acknowledged and more thoroughly researched problem than elderly drug abuse. Much of the literature on elderly alcohol abuse has been summarized (Gomberg 1980; Zimberg 1979; Schuckit & Pastor 1978).

Many researchers believe that the prevalence of elderly alcohol abuse is considerably lower than the level of alcohol abuse among other age groups. Cahalan, Cisin and Crossley (1969) found that older people have lower levels of alcohol consumption than younger adults, and some studies (e.g., Gorwitz et al. 1970) have reported that, at least for alcoholic patients in treatment facilities, the age incidence of alcoholism peaks between the ages of 35 and 50 years. Some reports espouse a sort of "maturing out" hypothesis for alcoholism and suggest that, at least for some individuals, alcoholism may be a self-limiting problem which is autonomously mitigated and controlled or entirely overcome in some older persons. However, other reports indicate that alcohol abuse among the elderly is in fact a serious problem which is underdiagnosed and underreported.

Bailey, Haberman & Alksne (1965), for example, in a household prevalence survey of 3,959 dwelling units in the Washington Heights area of New York City found that there was a second peak prevalence of alcoholism in the 65- to 74-year-old age group which was almost as high as the level of the first peak prevalence they found in the 45- to 54-year-old group. Interestingly, the prevalence among the 55- to 65-year-old age group was markedly lower than either of the two adjacent age groups. This decline in the prevalence from the 45-54 age group to the 55-65 age group may account for the belief of many researchers that alcoholism decreases with age. This would be reinforced by the failure of many surveys to include subjects over 60 years of age.

The actual prevalence of alcoholism among the general elderly population is unknown although estimates of elderly alcoholism based on limited studies range from 2-10 percent with estimated prevalences being considerably higher for elderly individuals who are widowers, nursing-home residents, patients of general medical wards and psychiatric patients (Zimberg 1979; Schuckit & Miller 1976). Diagnosis of alcoholism in the elderly is complicated, as the alcohol problem is likely to be denied and hidden by the elderly and often even by their families; elderly drinkers may even be unaware that they have an alcohol problem and they may suffer from symptoms which they do not realize are related to their drinking. Physicians are often unwilling or unable to recognize elderly alcohol abuse, and care facilities may not address it as a problem (Eckhardt 1978; Shropshire 1975; Zimberg 1974).

One important question which has been raised is whether or not elderly alcoholics developed their alcohol problems during old age or earlier in their lives. While there are probably four or five different patterns of alcoholism onset and reinitiation which describe elderly alcohol abuse, a number of studies have simply divided elderly alcoholics into two groups defined by early or late onset of the problem. These studies tend to concur with the finding that approximately two-thirds of the elderly alcoholics are "early-onset" alcoholics with longstanding problems while one-third are "late-onset" alcoholics who developed their drinking problems later in life. One of these studies (Rosin & Glatt 1971) found that late-onset alcoholism was associated with the stresses and problems of aging including depression, bereavement, retirement, loneliness, marital stress and physical stress. Late-onset alcoholics did not seem to have the deep-rooted psychological problems or the personality characteristics of younger alcoholics. These late-onset drinkers seem to use alcohol as a coping mechanism to adapt to the problems of aged individuals. Guttmann (1977) in a study of legally available drug use by 447 noninstitutionalized elderly people in the Washington, D.C. area found that 56.2 percent of the subjects reported little or no use of alcohol, 24.6 percent of the subjects used alcohol a few times a week to a few times a month and 18.6 percent of the respondents used alcohol daily. When asked about their reasons for using alcohol, 29.9 percent said that they used alcohol to forget about personal problems, 27 percent said they used it to "have fun," 22 percent said it was to be accepted by friends and two percent said it was to sleep better. 1.8 percent of the users admitted to having an alcohol habit and 1.1 percent admitted to having problems in the past year related to alcohol use. Elderly alcoholics generally consume less alcohol (per drinking occasion) than younger alcoholics but are more likely to drink on a daily basis (e.g., Schuckit & Pastor 1978, Harford 1977). These findings indicate that the elderly alcoholic has a different drinking pattern than his younger counterpart, the elderly alcoholic's pattern is in keeping with the idea that they are more likely to drink as a means of coping with the stresses of aging. According to Zimberg (1978a) the elderly alcoholic seldom engages in more than mildly severe drinking with acute intoxication no more than once per month. Although there may be some impairment in social interactions, family relations or occupational functioning, no severe physical or legal problems are likely, and the signs and symptoms of the alcohol-addiction pattern typical of younger alcoholics is less evident.

It is likely that the problem of alcohol abuse among the elderly, although serious, is less severe than the problem among younger age groups. However, the population at risk is proportionately smaller for the elderly. These individuals who are presently older grew up in cultural environments which for many included a strong temperance tradition. National surveys (e.g., Armor et al. 1977) show that the percentage of abstainers among the elderly to be 52 percent of the men and 68 percent of the women and that these percentages drop with each successively younger age group. While this may be due partly to a developmental trend of increased abstention with age, it is likely that this trend is partly a cultural phenomenon which means that the level of abstention of the currently old is greater than the level of abstention that is likely to be maintained by the next younger group (who are currently 50-64) when they reach their old age. This suggests that the percentage of users of alcohol in the 65 and older group will increase for the next 10 to 15 years. While an increase in the percentage of users does not necessarily mean an increase in the percentage of abusers, it does mean an increase in the population at risk and, unfortunately, it seems likely that there will be some increase in the magnitude of abuse; increase in per capita consumption of alcohol has been related to an increase in alcoholism (de Lint 1974, Faris 1974).

Other factors suggest problematic trends which may relate to increased elderly alcoholism over the next few decades. According to population projections by the U.S. Bureau of the Census (1977), as of 1980 the elderly should constitute 11.2 percent of the population in this country. They are expected to increase proportionately for the next 15 to 20 years, reaching 12.4 percent of the population by 1995. With the "aging of the population" more and more elderly people will be drawing on increasingly limited financial and institutional support systems. These systems are barely, if at all, able to meet current demands and the projected economic outlook suggests proportionately decreased rather than increased resources. This will mean increased stress for the elderly and, since external stress has been related to increased elderly alcoholism, these social trends might lead to increased elderly alcoholism.

Another trend which is troublesome is the acceptance of the idea that the use of alcohol is a reasonable problem-solving mechanism for the elderly. It seems that it is considered acceptable for the elderly to drink, but inconceivable that they might have drinking problems. In fact, common attitudes seem to be expressed in sentiments such as "what else have they got to live for," "it helps them – it's like medicine" and "at least when they've had a drink or two they're not so difficult." Some nursing homes and care facilities have even started providing alcohol in "beer and wine therapies" to "increase social participation" by the elderly patients (e.g., Becker & Conn 1978). No other population with interpersonal problems would be offered alcohol as a solution, yet the tendency to turn to pharmacological solutions for almost any problem an elderly person has seems to be accepted by many of the elderly and by many of the people who care for the elderly.

A fairly consistent picture of elderly alcoholism emerges from the literature. Elderly alcohol abuse is a significant problem; even the most conservative estimates would implicate a very large number of people. One-third of the elderly alcoholics developed their problems after entering their elderly years and their drinking seems to be related to attempts to cope with the stresses and problems of old age rather than to more deeply rooted psychological difficulties. Elderly alcohol abusers are likely to drink more often though in smaller quantities than their younger counterparts and their drinking is less likely to result in severe and obvious social or physical problems or impairments. Elderly alcoholism is often hidden, denied and unrecognized; the elderly abuser may even be unaware that his or her drinking is a problem. As the environmental stresses will increase for the elderly over the next decade and as a greater percentage of the elderly are at risk as nonabstainers, the number of elderly alcoholics may increase dramatically over the next 10 to 15 years.

A SYNTHETIC MODEL OF ELDERLY DRUG ABUSE

Elderly drug abuse may follow a pattern which is comparable to the pattern of elderly alcohol abuse and, by synthesizing some of the findings from research in these two areas, it is possible to develop a model of elderly drug abuse. Some of the findings about elderly drug abuse correspond to findings about elderly alcoholism. Both elderly alcohol abusers and elderly drug abusers do not seem to "mature out." There is likely to be an aging of both abuser populations and both these populations may reduce, adapt and/or conceal their abuse as they reach old age. Both forms of abuse may also begin or may reinitiate during old age. Elderly alcoholism and elderly drug abuse seem primarily to involve legal substances taken in small but frequent doses.

Since such a large percentage of the elderly take alcohol in combination with other drugs, it may well be that for many of the elderly both alcohol abuse and other drug abuse are part of a single pattern. Therefore, any increase in elderly alcohol abuse might effectively mean an increase in elderly drug abuse, and indeed, an increase in elderly alcohol abuse is expected over the next several decades. Adults who are now 45-49 years of age are not only less likely to be abstainers than the older adults, but are also much more likely to be heavy drinkers. While the level of drinking of the age group may decline to some extent as they grow older, it seems very likely that their drinking levels will not decline to the levels of those who are contemporarily older. Therefore, over the next few decades there is likely to be an increase in the number of elderly drinkers and an increase in the amount that they drink. This may well mean an increase in other drug abuse by the elderly, if the elderly do not decrease their use of psychoactive drugs. As the percentage of the elderly who abstain from alcohol is expected to decline over the next few decades, so might the reluctance among some of the elderly to use psychoactive drugs and the unwillingness of many to condone and to participate in the abusive use of these drugs. In the younger age groups there are already higher levels of drug use and abuse, and as these people age, they may not only become elderly abusers, but they may also change the attitudes of other elderly people about the acceptability of drug abuse.

It is reasonable to hypothesize that other aspects of elderly drug abuse will be found to correspond with some other findings about elderly alcohol abuse. Elderly drug abuse, like elderly alcohol abuse, is likely to be hidden, denied and unrecognized; the elderly abuser may not even recognize that he or she has a drug abuse problem. The pattern of drug abuse which the elderly adult is likely to engage in may well not produce severe, obvious social or physical disruptions or impairments. The abused substance or substances may be used almost as if they were medicines to treat the problem of old age; elderly abusers may think of their substance abuse as self-medication. Elderly abusers may use alcohol and/or other drugs as coping mechanisms which they believe are sanctioned by those around them. Substance use may substitute for alternative coping mechanisms, and the elderly substance abuser may become increasingly dependent on psychoactive drugs including alcohol. The initiation of alcohol abuse later in life has been associated with the difficulties of old age including physical problems, retirement, bereavement, marital stress, depression, loneliness and would be expected to increase as environmental stresses increase. Likewise, it seems probable that the initiation of drug abuse later in life would also be associated with these types of coping problems and would also increase in response to greater stress. The hypothesized model of elderly drug abuse, particularly those factors which may be antecedents of late-onset elderly drug abuse, can be supplemented with inferences from the literature on adolescent drug abuse.

ADOLESCENT DRUG ABUSE

There are a number of similarities between adolescence and old age. Both involve uncertain and changing roles and self concepts, lower social status, employment and income disadvantages, shifting and uncertain social supports, general attributions of inferior abilities and resources and, to some extent, estrangement from society. Many adolescents and many of the elderly share feelings of depression, loneliness, anxiety, confusion, conflicts between dependence and independence and an externally attributed locus of control. Of particular importance is that both groups are limited in their ability to become self-reliant and assert their independence. In addition, they both have limited resources for coping and both groups find themselves in difficult periods of transition. While there are certainly many major differences between these two groups, the similarities may be important in that they may involve some of those factors which have been discussed as antecedents of drug abuse. In addition, both groups seem to have readily available sources of drugs and both groups have their primary social interactions with people who condone heavy substance use. There is a fairly well-documented body of literature on the factors antecedent to adolescent drug abuse and, since some of these factors are present for the elderly, they may be predictive of elderly drug abuse.

Kandel (1978) in her review of some of the findings of longitudinal studies of drug abuse in normal populations summarizes reports of some of the antecedents of drug use which have been identified for adolescents. Some of these factors seem to be relevant to both adolescents and the elderly. These include an emphasis on dependence, depression, alienation, apathy, pessimism, anxiety, low self-esteem or a decrease in self-esteem, lower aspirations and motivations, lower levels of achievement, a positive attitude toward drug use among one's peers and a social setting favorable to drug use. These factors, many of which are related to coping problems, may constitute at least some of the antecedents of elderly drug abuse. Some of the identified antecedents of adolescent drug abuse are not commonly associated with elderly populations. These would include participation in delinquent and deviant activities, rebelliousness or the demonstration of attitudes and values which are favorable to deviance and which reflect lessened conformity to social institutions. These factors which are characteristic of adolescents but not of the aged may account, at least to some extent, for the observation that adolescents are heavy users of illicit drugs while the elderly are likely to use only licit substances. One other relevant report from the literature is the finding by Jessor, Jessor and Finney (1973) that the onset of marijuana use is not only associated with a set of psychosocial variables but is also associated with systematic changes in those same psychosocial variables. This suggests that individuals may be particularly prone to abuse drugs during periods of stressful transitions, and this is likely to include the transition to old age.

Also part of the literature on younger drug abusers is the highly controversial "stepping-stone" theory of drug addiction which hypothesizes that marijuana use leads to the use of "hard drugs" such as heroin (O'Donnell & Clayton 1981; Kandel & Faust 1975). It is generally accepted that hard drug use is typically preceded by marijuana use and that marijuana use is typically preceded by the use of alcohol and tobacco. The controversy centers on whether or not the earlier use of the softer substances causes the later abuse of the harder substances. Without attempting to resolve this controversy, it seems safe to say that the earlier substance use probably facilitates or makes at least some contribution to the later abusive substance use. This implies that even the licit use of licit psychoactive substances increases the risk of abuse. Additionally, it also seems likely that the greater the use of those earlier softer substances, the greater the increment in the risk of substance abuse. This suggests that the high use of psychoactive substances by the elderly may well increase their risk or their potential for drug abuse. Furthermore, it is currently believed by some researchers that some physicians and some care facilities overprescribe psychoactive medications (e.g., Petersen, Whittington & Bear 1979). If this is true, then many elderly and late middle-aged adults are using overly high levels of these drugs and they may come to rely on them as coping mechanisms. There is currently a movement to educate physicians in proper geriatric practices which is likely to lead to a decrease in the level of psychoactive drugs which are prescribed. This may in turn lead, over the next decade, to the creation of a group of elderly who suddenly cannot licitly obtain the high level of drugs on which they have come to rely. This may further increase the risk that at least some elderly drug users will turn to some form of drug abuse. If it is assumed that the earlier nonabusive use of psychoactive substances becomes incorporated into the set of coping mechanisms of the user, then the increment in the risk of later drug abuse may be reinforced for the elderly by certain developmental changes which seem to occur with aging.

PSYCHOLOGICAL CHARACTERISTICS OF THE AGED

The risk of drug dependence is increased for the aged by some of the psychological characteristics which are common among elderly adults. In addition to the decrease in physical abilities which accompanies aging in later life, it appears that there is some decrement in intellectual and problem-solving abilities (Botwinick

1978; Rabbit 1977) which, together with the reduced interpersonal supports available to many of the elderly (Lowenthal & Robinson 1976) may lead to a reduction in the coping abilities of many elderly adults. This may increase the tendency of some of the elderly to rely on drugs as problem-solving mechanisms. It also seems that with aging many of the elderly return to the use of more primitive defense mechanisms (Pfeiffer 1977), some of which would reinforce a reliance on drugs. Somatization, for example, is a defense which operates through a hypochondriacal preoccupation with medical complaints. Focusing on physical medical problems leads to the expectation of remediation in the medical realm which usually involves medication. As the physical complaint is likely to be groundless, untreatable or a problem which is greatly exaggerated by the somatizer, the physician may prescribe a psychoactive drug. This means that those elderly who do somatize will be motivated to acquire and use drugs, and the use of drugs may become established as a coping mechanism. Other primitive mechanisms such as unmodified anxiety and depression withdrawal might lead to the use of psychoactive substances which may be used to relieve these severe affective states; mechanisms such as denial and projection might prevent an elderly abuser from being aware of his or her abusive drug dependence. Neugarten and her associates (1964) have found consistent developmental differences involving a change with age from active to passive mastery styles of coping with the environment and a change from an outer world to an inner self-oriented focus. Psychoactive drug use is a relatively passive coping behavior which acts on and changes, at least temporarily, the user rather than the user's environment or situation. As such, it may be compatible with the more passive coping styles of many older adults. While none of the developmental changes in defense mechanisms, style of mastery or focus would be expected to cause drug abuse in the elderly, they seem likely to reinforce a reliance on drugs as a coping mechanism which may ultimately contribute to drug abuse.

CONCLUSION

The elderly demonstrate very high levels of drug use and they seem to be at high risk for drug misuse. There are indications in the literature that the elderly are susceptible to drug abuse as well as indications that this may be an increasingly serious problem for the aged over the next two decades.

The literature on elderly drug use, misuse and abuse is extremely limited. The increasing public concern for the elderly and the growing awareness of researchers of the potential problem of drug abuse by the aged have begun to stimulate research in this area. However, as this research is still in its infancy, it will be a number of years before there is substantial research-documented literature in this area. By synthesizing the available information about elderly drug use, misuse and abuse with the information which is available on elderly alcohol abuse, and by supplementing this synthesis with findings from research on adolescent drug abuse and gerontology, it was possible to formulate a hypothetical model of elderly drug abuse.

This model makes a prediction about the most probable pattern of elderly drug abuse, identifies some of the factors which might contribute to drug dependence and extrapolates a number of variables which are likely to be antecedent to drug abuse among the aged. The model hypothesizes that elderly drug abuse is related to coping problems. It suggests that there are a number of social and psychological factors which contribute to the development by some elderly adults of a reliance on psychoactive substances as a coping mechanism to deal with the stresses and problems of old age. These include such potential problems as retirement, physical problems, loss of family and friends, dependence, as well as feelings of depression, loneliness, anxiety, low self-esteem and alienation. The use of drugs as a coping mechanism may lead to drug abuse. The model proposes that the abuse is likely to involve small though relatively frequent doses of multiple legal substances taken in combination which may not result in severe obvious physical or social problems. The abuse pattern may be hidden, denied or unrecognized. Drug abusing adults may think of their substance use as self-medication and may even be unaware that their use of drugs is abusive.

The hypothesized model may be of some use in the development and direction of research on elderly drug abuse and may suggest an approach to the prevention of drug abuse among the aged. Elderly alcoholism has been shown to be related to the stresses of old age and elderly abuse of other drugs is also assumed to be related to these problems. A number of researchers (e.g., Zimberg 1978b) have suggested that the most effective treatment for elderly alcoholism is the improvement of the social and environmental circumstances of the elderly. This approach may also succeed with the elderly drug abuser. Taking this approach a step further, alleviating some of the problems of old age may also help to prevent substance abuse among the elderly.

There is currently little public awareness that drug abuse may be a serious problem for the elderly. It has been predicted that the stresses of old age will increase over the next two decades and that this will result in increased elderly drug abuse. Our society has devoted considerable resources to the study and prevention of drug abuse among the young. Just as our society is concerned with the future of the young, so should we be concerned with the future of the elderly.

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Medicine

COVER STORY

Out in the Open

Changing attitudes and new research give fresh hope to alcoholics

Just before the Betty Ford Center opened in the affluent desert town of Rancho Mirage, Calif., in 1982, neighbors ventured out across their well-manicured lawns to ask the staff a few questions. "Will there be bars on the windows?" they wanted to know. "Will they get out and go drinking in the neighborhood?" The answer in each case was of course no, but the questions reveal a familiar attitude toward alcoholics: many people thought of them as hardly better than criminals or at the very least disturbed and bothersome people. But at the same time the fact that a sanatorium for alcoholics had been started by a former First Lady who openly admitted to a drinking problem signaled that a hopeful change was in the air. Since then, a stream of recovering alcoholics, among them such celebrities as Elizabeth Taylor, Jason Robards and Liza Minnelli, have stepped forward to tell their stories with bracing candor—of being caught in the vortex of alcoholism, of taking the strenuous route to sobriety offered in therapy and of regaining their health and self-respect. The long process of recovering from alcohol abuse, which experts insist never ends, suddenly began to get favorable notices.

Today, in treatment centers nationwide, patients are getting a message of openness and hope. In his therapy sessions, John Wallace, director of treatment at Edgehill Newport, a center in Newport, R.I., explains that alcoholism is a disease with a genetic basis, and nothing to be ashamed of. "I ask how many had a close alcoholic relative," he says, "and 95% raise their hands. That astonishes them." He describes the latest theories about neurochemical imbalances that make an alcoholic incapable of drinking normally. "They are really fascinated," he says. "It takes away a lot of their guilt and makes them less defensive."

In ways unimagined ten years ago, the shadow that has obscured the truth about

[illegible]

alcohol has begun to lift. There is encouraging news, and it is substantial. "Silence is each day giving way to courage," Otis Bowen, Secretary of Health and Human Services, said recently, "and shame to strength." Evident all around is a busy sense of awakening. Children are learning about the perils of alcohol in school through slogans like "Get Smart: Don't Start—Just Say No." The accumulated scientific findings of the past decade are having a major impact on the public. Recently a Gallup poll found that a great majority of American adults are convinced that alcoholism is indeed an illness rather than a r of moral backsliding. In that, they e the support of the American Medical Association, which 21 years ago formally declared alcoholism a disease. At that time, only a handful of programs, such as Hazelden in Minnesota, offered treatment for alcoholics. Since then medical centers and treatment programs have proliferated across the country. There are more than 000 treatment programs, a 65% increase in the past six years alone. Partly because of the new spotlight on the dangers of alcohol, Americans are beginning to moderate their drinking habits: consumption of alcohol peaked in 1981 and has since declined by 5%. In many social circles today, the big drinker stands out like W.C. Fields at a temperance meeting.

The most exciting developments in the battle against alcoholism are taking place in the nation's laboratories, where scientists and medical researchers are probing its neurochemical roots and hunting for genes that may influence its development. Next month researchers from six national laboratories will meet in New York City to coordinate their search through human DNA for the genes that may underlie alcoholism. If they are successful, doctors may one day be able to test young people for certain genetic markers, the chromosomal quirks that predispose some individuals to alcoholism, and warn those who are at risk of developing the disease. Says Henri Begleiter, professor of psychiatry at the State University of New York Health Science Center and president of the Research Society on Alcoholism: "Never in the history of alcoholism have we made as much progress as we have in recent years."

For the 18 million Americans with serious drinking problems, life is a runaway roller coaster that, left untended, inevitably leads to disaster. "It ruins everything that matters to you," says New York *Times* Reporter Nan Robertson, a recovered alcoholic. "In the end, the bottle is your only friend. Alcoholics would rather do anything than stop drinking." For the vast majority of Americans, the occasional social drink is a harmless affair. For the afflicted, however, the most innocent gathering of family or friends—a wedding at a suburban country club, a casual gathering on an urban sidewalk—can turn into a nightmare of temptation, indulgence and worse. Recalls a youthful recovering alcoholic: "My biggest fear was getting through life without a drink. Today it is that I might pick up that one sucker drink."

The stakes are high. Alcoholism claims tens of thousands of lives each year, ruins untold numbers of families and costs $117 billion a year in everything from medical bills to lost workdays. The magnitude of the problem has been overshadowed in recent years by the national preoccupation with the new threat of AIDS and the widespread use of drugs such as heroin, cocaine, marijuana and crack. "Take the deaths from every other abused drug," says Loran Archer, deputy director of the National Institute on Alcohol Abuse and Alcoholism (NIAAA) in Washington. "Add them together, and they still don't equal the deaths or the cost to society of alcohol alone."

Alcoholism's toll is frightening. Cirrhosis of the liver kills at least 14,000 alcoholics a year. Drunk drivers were responsible for approximately half the 46,000 driving fatalities in the U.S. in 1986. Alcohol was implicated in up to 70% of the 4,000 drowning deaths last year and in about 30% of the nearly 30,000 suicides. A Department of Justice survey estimates that nearly a third of the nation's 523,000 state-prison inmates drank heavily before committing rapes, burglaries and assaults.

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As many as 45% of the country's more than 250,000 homeless are alcoholics.

Despite all the advances in knowledge and attitudes, plus the deluge of books, movies and television programs on alcoholism, the cartoon image of the cross-eyed drunk slumped in the gutter or staggering through the front door still lingers in the minds of some Americans. Not long ago many believed, as two researchers put it in the 1950s, that "alcoholism is no more a disease than thieving or lynching." Such attitudes are fading fast, to be sure, but not without leaving a residue of ambivalence. Says LeClair Bissell, 59, a recovered alcoholic and physician: "At the same time we say through our lips that alcoholism is a chronic disease, many of us feel in our guts that it's a moral or self-inflicted problem."

Yet it is a disease, and it can be a ruinously expensive one. A four-week drying-out regimen can cost anywhere from $4,000 to $20,000 for in-patient care; today medical insurance covers the tab for 70% of American workers in companies with more than 100 employees. In the early 1970s, the Kemper Group of Long Grove, Ill., was the first national insurance company to include coverage for alcoholism in all its group policies. The firm's hunch: the bill for helping an alcoholic quit today would be cheaper than nursing him through afflictions like cirrhosis of the liver and strokes later in life. The logic of acting sooner rather than later has since spread throughout corporate America. Some 10,000 firms and public agencies, including 70% of the FORTUNE 500 companies, now have employee-assistance programs to help alcohol and drug abusers pull their lives together and get back to work. "Before this," says William Durkin, employee assistant manager at ARCO, "the normal handling was to tolerate the alcoholic employee until he became intolerable and then to fire him."

Progress in the actual treatment of alcoholism is disappointing. Most facilities still rely on basic therapies worked out in the 1940s. Though some centers advertise grossly exaggerated success rates of 70% after four years, the best estimates are that only 12% to 25% of patients manage to stay on the wagon for three years. Alcoholics Anonymous, the tremendously popular association of an estimated 1 million recovering alcoholics, remains the single biggest source of support for chronic drinkers. But its record is hard to assess because of members' anonymity. Even so, only 15% to 20% of alcoholics get any treatment at all. Says Enoch Gordis, director of the NIAAA: "Something very important is still missing here."

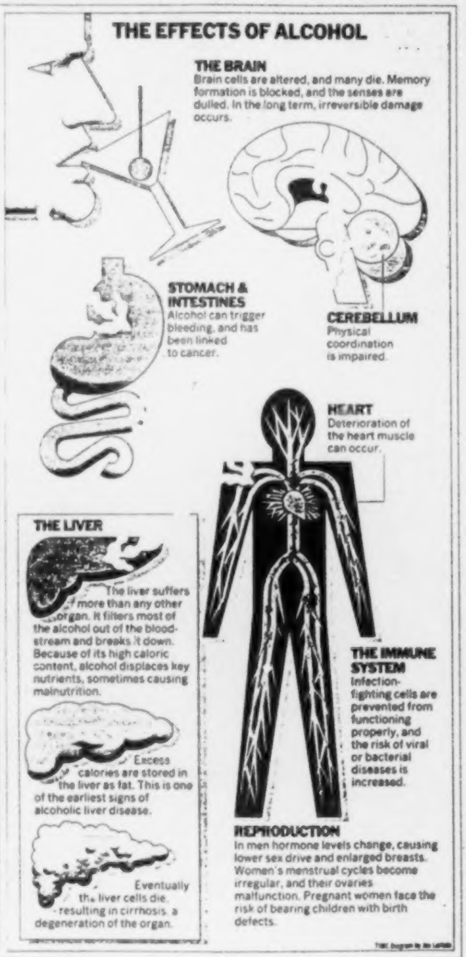
Simultaneously, another shadowy fact of life about alcoholics has been dragged into the light: the severe emotional scars they leave on their spouses and especially on their children. "Years ago the focus fell solely on the alcoholic," says Carol, a mother of four and wife of an alcoholic. "Nobody identified the needs of the family." Indeed, alcohol abuse accounts for more family troubles than any other single factor. A Gallup poll this year found that one in four families reported a problem with liquor at home, the highest reported rate since 1950 and twice the 1974 rate. According to Health Secretary Bowen, alcohol is the culprit in 40% of family-court cases and accounts for between 25% and 50% of violence between spouses and a third of child-molestation incidents.

Though awareness of alcoholism's de- ctiveness is growing, the sheer number lcoholics shows no sign of abating. g people are especially vulnerable. n states that nearly 5 million adoles- or three in every ten, have drinking problems. Several studies show that children are beginning to drink earlier than ever before, and a *Weekly Reader* study ier this year reported that 36% of th-graders were pressured by peers to k. "Kids are making decisions about ohol and drugs when they are 12 to 14, ereas in the preceding generation they ade those decisions at ages 16 to 18," says e Dogoloff, executive director of the merican Council for Drug Education. The younger a person starts drinking, the ore likely he is to develop problems later life."

Who, exactly, is an alcoholic? The question is a tricky one: symptoms are not always clear cut, and even doctors do not agree on a definition of the disease. The extreme cases are obvious. A person in the grip of alcoholism blacks out from drinking too much, suffers memory loss, and wakes up trembling with craving for another drink. But most cases show fewer dramatic symptoms. Also, the behavior of alcoholics fluctuates wildly. Some drink heavily every day, while others can stop for brief periods, only to go off on binges. This past year the American Psychiatric Association settled on three basic criteria to define and diagnose alcoholism: physiological symptoms, such as hand tremors and blackouts; psychological difficulties, which include an obsessive desire to drink; and behavioral problems that disrupt social or work life.

The search for alcoholism's genetic underpinnings began in earnest in the early 1970s with a simple question: Why does the disease seem to run in families? Dr. Donald Goodwin, chairman of the psychiatry department at the University of Kansas School of Medicine, set about seeking an answer by studying 133 Danish men who were all adopted as small children and raised by nonalcoholics. Goodwin divided his subjects into two categories: those with nonalcoholic biological parents and those with at least one alcoholic parent. Then he interviewed each of the adopted men in depth and examined health records to see which of them developed alcoholism in adulthood. If the disease had a genetic basis, Goodwin reasoned, then the children who had an alcoholic biological parent would wind up with drinking problems more often than the others.

His findings were startling. The sons



THE EFFECTS OF ALCOHOL

THE BRAIN
Brain cells are altered, and many die. Memory formation is blocked, and the senses are dulled. In the long term, irreversible damage occurs.

STOMACH & INTESTINES
Alcohol can trigger bleeding, and has been linked to cancer.

CEREBELLUM
Physical coordination is impaired.

HEART
Deterioration of the heart muscle can occur.

THE LIVER
The liver suffers more than any other organ. It filters most of the alcohol out of the bloodstream and breaks it down. Because of its high caloric content, alcohol displaces key nutrients, sometimes causing malnutrition.

Excess calories are stored in the liver as fat. This is one of the earliest signs of alcoholic liver disease.

Eventually the liver cells die, resulting in cirrhosis, a degeneration of the organ.

THE IMMUNE SYSTEM
Infection-fighting cells are prevented from functioning properly, and the risk of viral or bacterial diseases is increased.

REPRODUCTION
In men hormone levels change, causing lower sex drive and enlarged breasts. Women's menstrual cycles become irregular, and their ovaries malfunction. Pregnant women face the risk of bearing children with birth defects.

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of alcoholics turned up with drinking problems four times as often as the sons of nonalcoholics. That result helped put to rest the popular assumption that alcoholics took up drinking simply because they learned it at home or turned to it because of abuse suffered at the hands of an alcoholic parent. The study, however, did not rule out environmental factors. Indeed, scientists now estimate that fully 30% of alcoholics have no family history of the disease. But Goodwin showed that some inherited attribute was involved. "What we learned from the adoption studies," says Dr. C. Robert Cloninger, a professor of psychiatry at Washington University in St. Louis, "is not that nature was important or nurture was important but that both are important."

But it was still far from clear how hereditary and environmental factors combine to create an alcoholic. In the early 1980s, Cloninger joined a team of Swedish investigators led by Michael Bohman, a psychiatrist at the University of Umeå, to study an even larger group of adoptees. Since Sweden's extensive welfare system keeps thorough records on each citizen, Bohman was able to compile detailed sketches of 1,775 adopted men and women, more than a third of whom had an alcoholic biological parent. As Cloninger studied the health, insurance, work and police records of his subjects, two distinct categories seemed to emerge—and with them new evidence that alcoholism may have more than one form.

Cloninger's first group of alcoholics, about 25% of the total, tended to drink heavily before the age of 25, had bad work and police records and met with little success in treatment programs. Drinking was a habit they seemed to pick up on their own, with little encouragement from friends or other influences. When Cloninger checked how often alcoholism appeared in the sons of men who fit this description, he found it surfaced nine times as often as in the general population. This variation of the disease, Cloninger concludes, is heavily influenced by heredity. Because it appears primarily in men, he calls this form "male limited" alcoholism.

The second type included both men and women and made up about 75% of the study's alcoholics. They started chronic drinking usually well after the age of 25, rarely had trouble with the law, and often successfully kicked the habit. Their children were only twice as likely to have trouble with alcohol compared with the general population. Cloninger labeled this category of alcoholism "milieu limited."

Diary of a Drunk

What is it like to suffer from alcoholism? The writer of the following article, who spent 28 days in a treatment center in the Northeastern U.S., offers his reflections.

Dying of alcoholism normally takes years. But before a final, prolonged bout of uncontrolled drinking caused my physical collapse and led to treatment, there was no doubt I was well on my way. My appearance was shocking. I was about 20 lbs. underweight and malnourished, the result of giving up almost all forms of food except coffee, sugar and, of course, alcohol. I was in the early stages of delirium tremens, the DTs. I sometimes heard faint ringing noises in my ears and suffered unexpected waves of vertigo. I felt near constant pressure in my lower back and sides from the punishment my liver and kidneys were taking. My personality was also seriously diseased. I was nervous, reclusive, by turns extravagantly arrogant and cringingly apologetic. I tried to cover my extremes of mood with brittle cheerfulness, even though I was desperately afraid. If you asked me how I was feeling, I usually lied. "Just fine."

I now see "just fine" as a key phrase that encompassed my diseased physical and mental condition. At the nadir, my addiction to a chemical that was killing me was nearly complete. I knew that something was very wrong with me. I even knew I was an alcoholic, but I had long since come to believe there was nothing I could do about it. I had decided that it was perfectly appropriate—just fine—that I should die. In fact, I honestly hoped that I would, sparing further grief for many people I loved. Dying, I thought, was the best thing I could ever do for them. The idea of living without alcohol could not occur to me. I preferred the idea that I was a hopeless case.

No one finds alcoholism more mysterious than the suffering alcoholic, and I was no exception. I had no idea why I was an alcoholic at all, though I should have: my father was one. But from his illness I had gained only a morbid fear of the substance, which lasted until I reached college. I would never touch the stuff. That prolonged abstinence while my adolescent peers experimented with liquor only made what happened to me more mystifying. I thought I could take alcohol or leave it.

Why did I ever start to drink at all? The short answer is that initially it made me feel better. Alcohol numbed my self-awareness, the same trick that it performs for nonalcoholic drinkers at cocktail parties. The difference is that normal drinkers dull their self-consciousness only slightly, the better to socialize. I very quickly tried to send all my thoughts and feelings about myself to oblivion. Psychologically, I was undoubtedly depressed when I began to overcome my well-founded but ill-understood fears about alcohol: my father died when I was a sophomore. For whatever reason, I spent the better part of two decades trying to stay emotionally and physically numb.

Even in those early days, signs might have pointed an expert on alcoholism toward my growing problem. One hint was my immediate tendency to drink to unconsciousness. At parties, I would often fall asleep in mid-hullabaloo on the couch. That drew plenty of jokes at the time. Only much later did I recognize that I had been passing out. Another signal was an initial, abnormally high tolerance for alcohol, at least until the passing-out stage. I thought I could hold my liquor pretty well. Now I think it means that my body was being less dutiful than most in handling overdoses of a hazardous chemical. (Years later, when only a couple of drinks would overload my toxified liver, causing slurring of words and other drunken symptoms, I finally joined the company of those who "can't hold a drink.")

Exactly when did I become addicted to alcohol? I don't know that either. The addiction was preceded by a delusion. I thought I drank to socialize. Maybe I did. My alcoholism took years to develop into a chronic affliction, and during much of that time I went to bars after work, one of the guys. The delusion was gradually reinforced by gravitation. I mingled more and more with other persistent drinkers who took longer and longer to call for their bar tabs. Most of us were actually alcoholics in varying stages of development. The nonalcoholics had long ago

licating a genetic predisposition to the ease that is triggered by extended avy drinking.

Cloninger's work added key pieces to puzzle of alcoholism by suggesting its that certain types of alcoholics have common. For example, Cloninger found that his male-limited alcoholics tended to be aggressive, even violent types. He hypothesizes that the nervous system underlying such behavior may react to alcohol in a way that quickly leads to dependence. "It's not proved," says Cloninger. "It's testable." Says Boris Tabakoff of the NIAAA: "For those of us looking for biological markers, Dr. Cloninger's work gives us a road map we can follow to link genetic traits to behavior."

If researchers could develop medical tests that identify biochemical signposts indicating a predisposition to alcoholism, they could warn potential alcoholics before trouble started. SUNY's Begleiter found just such a potential marker in the brain. By using an electroencephalograph to measure the brain waves of nondrinking sons of alcoholic fathers, Begleiter discovered that a particular brain wave called the P_3 showed a dampened response. In each instance the sons' brain waves closely duplicated those of their fathers, while other subjects with no family history of alcoholism showed strong P_3 waves. In addition, Dr. Marc Schuckit, a researcher at the San Diego Veterans Administration, has found that after several drinks some men whose fathers are alcoholics show fewer changes in the levels of two hormones, prolactin and cortisol, than men whose fathers are nonalcoholics. Eventually, such findings may provide important clues in the search for the genes involved in alcoholism.

Scientists acknowledge that work on the effects of alcohol on individual brain cells is still in its infancy. Part of the problem is that ethanol, the active ingredient in alcoholic drinks, easily penetrates the membranes of all cells and disrupts their normal function. Unlike other psychoactive drugs, ethanol does not target specific parts of nerve cells, or neurons, but seems to enter cell membranes and sabotage the nervous system indiscriminately.

Steven Paul, chief of the clinical neuroscience branch at the National Institute of Mental Health, is studying how ethanol affects certain cells in the brain to induce sedative effects. He is looking at a group of receptors, sites on the membranes of brain cells, that link with a molecule called gamma-aminobutyric acid (GABA), a neurotransmitter that moves

elected themselves out. Those of us who remained agreed that e were "normal." Unhappy, but normal.

Alcoholic perception is like that, in a hundred insidious and istorting ways. All of them are aimed at protecting a drunkard's otion that he is possessed of free will. My drinking buddies and I agreed that we did not have a drinking problem. Everything in our increasingly narrow world, though, was a problem that required drinking: the wife, the kids, the boss, the government. In dingy watering holes from which everyone with a healthy life to lead had gone home, we conspired to overlook the obvious, that our bodily cells were addicted, and our minds were along for the ride.

Inexorably, the need for alcohol grew, while the lies wore thin. As my alcoholism accelerated, I abandoned most drinking partners and joined the ranks of solitary topers bellied up to countless bars. I lost any sense at all of what would happen after I started drinking. I became completely unpredictable. Sometimes I would go home after a couple of drinks (there was usually more booze there). More often, I would join the lineup of other alcoholics at the bar telephone stalls, fumbling with worn-out excuses about unexpected visitors and urgent business meetings. Sometimes I would simply hole up in my office with a bottle after everyone else had gone home. There simply wasn't anything else in my life. Most frightening of all, I began to suffer alcoholic blackouts during drinking episodes. I would swim back into consciousness with no recollection of where I had been or what I had done. Once, I came to late at night on a downtown city street with my suit trousers slashed down one side by a razor.

"My personality was seriously diseased. I was nervous, reclusive, by turns extravagantly arrogant and cringingly apologetic."

Bizarre incidents like that left me petrified but unable to stop drinking. None of the growing physical pangs of alcoholism—the retching, nervous spasms, sweaty and sleepless nights, dehydration—matched the moments of hammering panic I felt every morning for months on end, as I tried to remember exactly what I had done the night before. At one point, terrified that I might kill someone with my car, I gave up driving, but never alcohol. Along with the fear came sudden rages—at my wife, at my friends, at anyone who tried to stop me from drinking. My homelife became a nightmare. Creeping paranoia set in.

No one wanted me to stop drinking more than I did. What I could not say was that I did not know how to do it. Every day, the inability inspired waves of remorse and self-loathing. But in my fearfulness, I stayed willfully ignorant of alcoholism. I would walk out of the room if a television commercial mentioned the subject. I was convinced that getting sober was merely a matter of personal willpower—and that, through some unfixable flaws of character, I lacked the power. I never wanted to be reminded of what was, to my mind, a moral affliction. Who would?

I still consider the fact that I did not die to be a miracle, meaning that some kind of providence intervened. For me, it took the form of a friendly superior at work who confronted me. I finally broke down and admitted that I needed help. That simple admission, so long in coming, brought an enormous release. Suddenly, alcoholism was no longer something I had to endure in private. Somehow, in that encounter, a powerful psychosis dissolved.

Years later, after hundreds of Alcoholics Anonymous meetings and many hours of intensive counseling, I am happy to acknowledge that I have a serious, progressive ailment, with no cure. Alcohol is no longer a terrifying, destructive force in my life. It is just another chemical, fine for you, perhaps, but deadly for me. I avoid it, but without a sense of panic or fear. Friends say I am a completely different person now. Only, sometimes, I remember the feelings of hopelessness and shame from those terrible years, and I still have to struggle to hold back the tears.

across the synapses between neurons. GABA homes in on a complex known as the GABA-benzodiazepine receptor. If there are a sufficient number of GABA molecules present in certain areas of the brain, anxiety diminishes. Tranquilizers such as Valium and Librium work by attaching themselves to the receptor and increasing GABA's effectiveness.

Paul believes ethanol also reduces anxiety by acting on those GABA-sensitive neurons. Altering the amount of GABA in the brain could theoretically neutralize the effects of intoxication. To that end, Paul is currently experimenting with a drug, Ro15-4513, that blocks ethanol's ability to activate the GABA receptor, thus sharply reducing alcohol's sedative effects in rats. Although the drug is toxic to humans, variants could one day be useful in treatment. Other scientists are studying a new class of drugs that seem to block the alcoholic's craving for a drink. These compounds boost the amount of another neurotransmitter, serotonin, in the brain, thus encouraging a sense of well-being—and bolstering abstinence.

Ethanol has a harmful effect on nearly every organ in the body. Chronic heavy drinking increases the risk of myocardial disease and high blood pressure. Alcohol eats away at the stomach and intestines, causing bleeding in some drinkers. Alcoholic males may experience shrunken testes, reduced testosterone levels, and even impotence. Sustained drinking sometimes disrupts women's menstrual cycles and can render them infertile. Among expectant mothers, drinking can produce birth defects and is a major cause of mental retardation in American children. Even the immune system's efficiency is reduced by alcohol. Studies are under way to determine whether heavy drinking might cause AIDS to surface more quickly in infected carriers.

But alcohol takes the worst toll on the liver, where most of the ethanol in the bloodstream is broken down. Because alcohol is so high in calories (there are 110 calories per jigger of 90-proof liquor), the liver metabolizes it instead of important nutrients, a phenomenon that can lead to severe malnutrition. The high caloric content of ethanol also causes fat to build up in the liver, one of the earliest stages of alcoholic liver disease. This is frequently followed by scarring of the liver tissue, which interferes with the organ's task of filtering toxins from the blood. The slow poisoning leads to other complications, including cirrhosis, an often fatal degeneration of the liver that affects at least 10% of all alcoholics and is especially hard on women. "They die of cirrhosis earlier than men, even though they consume less alcohol," says Judith Gavaler, an epidemiologist at the University of Pittsburgh Medical School.

This year studies at the Harvard Medical School and the National Cancer Institute reported that even women who drink moderately may have a 30% to 50% greater chance than nondrinkers of developing breast cancer. Heavy drinking among men and women alike has been linked to cancer of the liver, lung, pancreas, colon and rectum. In October a team led by Dr. Charles Lieber, a leading alcoholism researcher at the Bronx Veterans Administration Medical Center in New York City, reported that it had isolated a possible link between alcohol and cancer in humans. The culprit appears to be a member of the family of enzymes called cytochrome P-450s. In the presence of alcohol, the cytochrome can turn certain chemicals in the body into carcinogens.

Despite the medical recognition of alcoholism as a disease 21 years ago, there is still uncertainty over its legal status as an illness. Michael Deaver, the former aide to President Reagan who is on trial for lying to a grand jury about his lobbying activities, is arguing that he was not responsible because he is an alcoholic and his drinking at the time impaired his memory of events and facts. In the past the so-called alcoholism defense generally has not been very successful, but it has worked on occasion in perjury cases.

Next month the Supreme Court will

hear a case that is likely to hinge on the Justices' decision as to whether alcoholism is a disease. Two former soldiers, now recovered alcoholics, are seeking to overturn a 56-year-old Veterans Administration policy that classifies alcoholism as "willful misconduct" rather than a sickness. The VA's definition prevents alcoholics from receiving benefit extensions awarded to veterans with illnesses. In seeking to make their case, the plaintiffs' lawyers are expected to bring up the new evidence that holism may have a genetic basis. Says Johnson, general counsel for the N.C.A., which filed an amicus brief in the case: "We want a medical judgment, not a ruling based on fear, misunderstanding and prejudice."

For alcoholics, the only way to stop the havoc alcohol causes is, of course, to quit drinking. That is easier said than done. The main barrier to ending the torment is the alcoholic's characteristic, and usually adamant, denial that any problem exists. Mary, 61, who has not taken a drink for 14 years, remembers blacking out and waking up with her hands trembling so badly that she could not hold a cup of coffee. "I had reasons for all those things happening to me," she says, "and none of them had to do with my drinking."

How, then, to break the psychological impasse? One way is to follow a strategy called intervention, which was pioneered in the early 1960s by Vernon Johnson, an Episcopal priest in a Minneapolis suburb. In intervention, family members, friends and co-workers directly confront the alcoholic to shatter his carefully nurtured self-delusions. Beforehand they meet with a specially trained counselor (the fee: $500 to $750) to rehearse. In the actual confrontation, the alcoholic is presented with a tough but sympathetic portrayal of the mess he is in and is urged to accept prearranged admission to a treatment center, often on the same day. Says Carol Remboldt, publications director at Johnson's institute in Minnesota: "Intervention allows a tiny aperture to be poked in the wall of an alcoholic."

The process can be painful. A 31-year-old daughter read her alcoholic parents a letter in which she described how she had seen her mother change "from the best friend I ever had" to an unhappy and unreliable woman. "The good parts of your character," she said, "are being stolen away by alcohol. Don't let that bottle overtake your life." Indeed, children often provide the most persuasive statements. One alcoholic's resistance crumbled when his son said, "Daddy, when you read me the funnies on Sunday morning, you smell." Peggi, a former schoolteacher and recovered alcoholic, remembers the day seven years ago when she was faced down by her husband, sister and three sons. "It was awful," she recalls. "But it was crucial for me to see how my drinking affected their lives."

As Poet Robert Bly, the son of an alcoholic, puts it in a book called *Family Secrets*, edited by Rachel V. (Harper & Row, 1987): "Every child of an alcoholic receives the knowledge that the bottle is more important to the parent than he or she is." To mend the damage from those year-in, year-out traumas, hundreds of thousands of Americans have turned to Al-Anon and other family-therapy organizations. An offshoot of A.A. that was formed in 1951 for relatives and friends of alcoholics, Al-Anon has more than doubled in size since 1975 and now boasts some 26,000 regional groups. But the real comer is the children-of-alcoholics movement, aimed at the nearly 30 million offspring of chronic drinkers in the U.S. Made up of a variety of organizations, the movement took off four years ago with the best-selling book *Adult Children of Alcoholics*, a guide to the dilemmas C.O.A.s face, by Janet Geringer Woititz, a human-relations counselor in Verona, N.J.

At a typical C.O.A. meeting, participants sit in a circle and offer reflections on their own experiences, from a paralyzing fear of intimacy to acute conditions like bulimia, a disorder marked by episodes of excessive eating. At the heart of their pain and confusion is a childhood fraught with anxiety. "When we were kids and our parents were drunk, it was our problem," a 21-year-old daughter of an alcoholic told TIME's Scott Brown. "Somehow it seemed that we should be super people and make our family healthy." Reliving painful childhood experiences among sympathetic listeners enables the C.O.A.s to feel emotions they had suppressed. Recalls Rokelle Lerner, a pioneer in the movement: "I had to learn to re-parent myself, to comfort the little girl inside."

For both family members and chronic drinkers, the greatest frustration is the absence of a surefire treatment for alcoholism. The truth is that success rates often depend more on the individual makeup of the alcoholic than on the treatment. Alcoholics fitting Cloninger's male-limited type are less likely to remain sober after treatment, along with those with unstable work and family backgrounds. "The best predictor of patient outcome is the patient," says Thomas Seessel, executive director of the National Council on Alcoholism. "Those who are steadily employed, married and in the upper middle class are more likely to succeed. They have more to lose." In response to allegations that some centers have exaggerated how well their patients do after treatment, Congress has ordered the NIAAA to investigate treatment programs.

Today about 95% of in-patient treatment centers in the U.S. use a 28-day drying-out program developed in 1949 at Hazelden. For the first few days, staff help patients through the tremors and anxiety of withdrawal. From that point on, the emphasis is on counseling. The aims: dispel the alcoholic's self-delusions about drinking, drive home an understanding of alcohol's destructive properties, and make it clear that the only reasonable course is

to stop drinking—permanently. Some centers use Antabuse, a drug that induces vomiting and other symptoms if the patient has a drink. Schick Shadel, a program with hospitals in California, Texas and Washington, employs aversion therapy to condition alcoholics to recoil at the smell, taste and even sight of a drink. Most programs, however, rely on A.A. or other counseling programs to help reinforce the message of abstinence.

"Everyone knows how to get sober," says Michael Baar, an Albany, Calif., psychologist. "The problem is keeping them in that state." Relapse prevention is the latest attempt to help reduce the number of recovering alcoholics who fall off the wagon. Terence Gorski, president of the Center for Applied Sciences in Hazel Crest, Ill., has studied thousands of relapse cases and found that on their way to recovery, alcoholics go through specific stages, each with its dangerous temptation to return to drinking. Early on, it may be hard to cope with withdrawal. Later, the patient may falter in developing a normal family and social life. Finally, there is a period of complacency, when the recovering alcoholic no longer fears drinking as he once did. At each point, says Gorski, "the person is out of control before he actually starts to drink." His solution: counselors who meet regularly with recovering alcoholics to help them identify and face problems before they get out of hand. Says Gorski: "It is compatible with A.A. and self-help groups. The only difference is that we go beyond what A.A. has to offer."

Will there ever be a simple cure for alcoholism? Probably not. Even so, the next decade or so holds dramatic promise for advances in understanding and effectively treating the disease. Researchers hope eventually to sort out alcoholics according to the neurochemical bases of their addiction and treat them accordingly. "We are still trying to map out these neurochemical systems," says Edgehill Newport's Wallace. "If we succeed, then it is likely that we will be able to design treatments." A.A. and other groups may always be necessary to help alcoholics assess the psychological and emotional damage of chronic drinking, but there is hope that medicine may make the course to sobriety less perilous.

Medical and scientific promise, however, should not eclipse the importance of public policy efforts to curb heavy drinking among adults—and stop it altogether among youngsters and adolescents. Education is one approach. The Government's "Be Smart" campaign, aimed at eight-to-twelve-year-olds, has had some success. Mothers Against Drunk Driving has been a primary factor in the fight that has raised drinking ages from 18 to 21 in 34 states plus the District of Columbia since 1982. Despite strong opposition from the alcohol industry, which lobbies vigorously against higher excise taxes for alcohol and warning labels on beer, wine and liquor bottles, groups like MADD and the National Council on Alcoholism continue to push initiatives that will further discourage consumption of alcohol.

In his speech two weeks ago, Health Secretary Bowen complained that brewers and beer distributors spend $15 million to $20 million a year marketing their products on college campuses, encouraging heavy drinking and "contributing to poor grades, excessive vandalism, many injuries, and not so infrequently, death." Bowen asked Education Secretary William Bennett to encourage university presidents to restrict alcohol promotions on campus. Spuds MacKenzie, the canine star of Anheuser-Busch's advertising campaign for Bud Light beer, is also in the doghouse. This fall the National Association of State Alcohol and Drug Abuse Directors filed complaints with several federal agencies charging that the campaign encouraged kids to drink.

For those who know what British Novelist Malcolm Lowry described as the alcoholic's "fine balance between the shakes of too little and the abyss of too much," sobriety cannot come too soon. That is the challenge for medical researchers. But just as much energy should go into the job of preventing the disease. That means not only finding genetic markers to warn those susceptible but also changing attitudes in a society that still glorifies drinking. As Bowen remarked recently, "To do anything less than all this would be a disservice to ourselves, our society and to the many future generations whose lives and livelihoods are at stake." For millions of American alcoholics, there is no time to lose. —*By Edward W. Desmond. Reported by Barbara Dolan/St. Louis, Andrea Dorfman/New York and Melissa Ludtke/Boston*

Alcohol and the Family

The children of problem drinkers are coming to grips with their feelings of fear, guilt and rage

Believe it or not, there are still people who think that the worst thing about drinking is a hangover.

Oh, yeah, on New Year's Day I had a hangover that . . .

No. Forget hangovers.

Huh? So what should we talk about? Cirrhosis?

If you wish, but the liver, with its amazing powers of regeneration, usually lasts longer than the spouse, who tends to fall apart relatively early in the drinker's decline.

You're making it hard for a man to drink in peace.

Sorry, but even if spouses do not abuse alcohol, they can come to resemble drunks, since their anger and fear are enormous: way beyond what you'd find in a truly sober person.

I know, I know, it's terrible what goes on behind closed doors.

You make it sound like there are no witnesses. You're forgetting the children. They grow up watching one out-of-control person trying to control another, and they don't know what "normal" is.

I suppose it's hard for the kids, until they move out.

They may move out, but they never leave their parents behind.

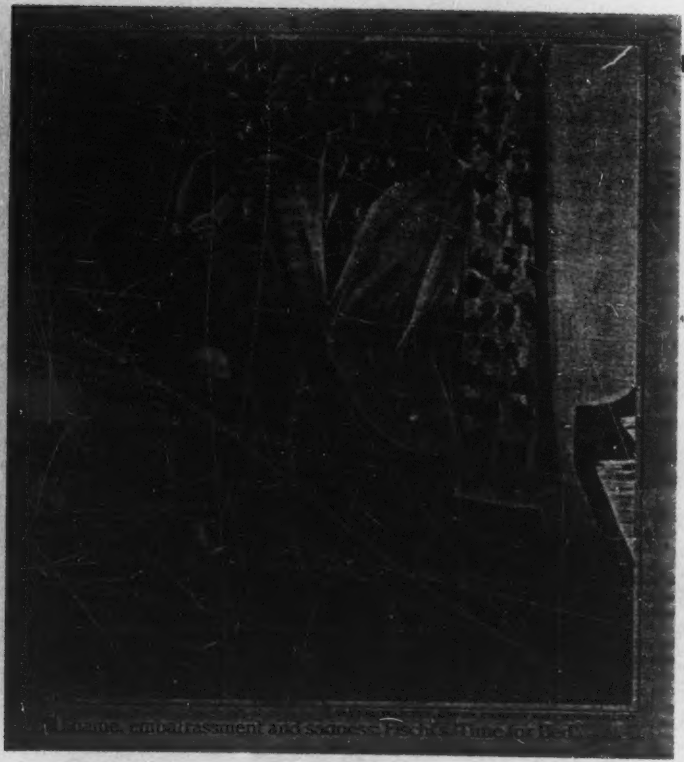
Hmm. Listen, can we talk?

We already are. A lot of people already are.

We are, just now, learning more about heavy drinking, and, simultaneously, putting behind us the notion that what alcoholism amounts to is just odd intervals of strange, and sometimes comic, behavior: W. C. Fields, Dean Martin, Foster Brooks. Since 1935 the members of Alcoholics Anonymous have been telling us, with awesome simplicity, that drinking made their lives unmanageable; Al-Anon brought us the news that relatives and friends of drinkers can suffer in harmony; and then came Alateen and even Alatot, where one picture of a stick person holding a beer can is worth a thousand slurred words. The Children of Alcoholics (COAs)—loosely organized but rapidly growing throughout the United States—reaffirm all of the previous grass-roots movements and bring us new insight into alcoholism's effects on the more than 28 million Americans who have seen at least one parent in the throes of the affliction. The bad news from COAs: alcohol is even more insidious than previously thought. The good news: with the right kind of help, the terrible damage it does to nonalcohol-

LEIF SKOOGFORS—WOODFIN CAMP

Exorcising old demons: *Gill (rear) undergoes grief therapy at Caron Family Services*



Shame, embarrassment and sadness: Fischl's 'Time for Bed'

ics need not be permanent.

Imagine a child who lives in a chaotic house, rides around with a drunk driver and has no one to talk to about the terror. Don't think it doesn't happen: more than 10 million people in the United States are addicted to alcohol, and most of them have children. "I grew up in a little Vietnam," says one child of an alcoholic. "I didn't know why I was there; I didn't know who the enemy was." Decades after their parents die, children of alcoholics can find it difficult to have intimate relationships ("You learn to trust no one") or experience joy ("I hid in the closet"). They are haunted—sometimes despite worldwide acclaim, as in the case of artist Eric Fischl—by a sense of failure for not having saved Mommy or Daddy from drink. And they are prone to marry alcoholics or other severely troubled people because, for one reason, they're willing to accept unacceptable behavior. Many, indeed, have become addicted to domestic turmoil.

'Hurting so bad': Children of alcoholics are people who've been robbed of their childhood—"I've seen five-year-olds running entire families," says Janet Geringer Woititz, one of the movement's founding mothers. Nevertheless, the children of alcoholics often display a kind of childish loyalty even when such loyalty is clearly undeserved. They have a nagging feeling that they are different from other people, Woititz points out, and that may be because, as some recent scientific studies show, they are. Brain scans done by Dr. Henri Begleiter of the State University of New York College of Medicine in Brooklyn reveal that COAs often have deficiencies in the areas of the brain associated with emotion and memory. In this sense and in several other ways—their often obsessive personalities, their tendency to have a poor self-image—the children of alcoholics closely resemble alcoholics. In fact, one in four becomes an alcoholic, as compared with one in 10 out of the general population.

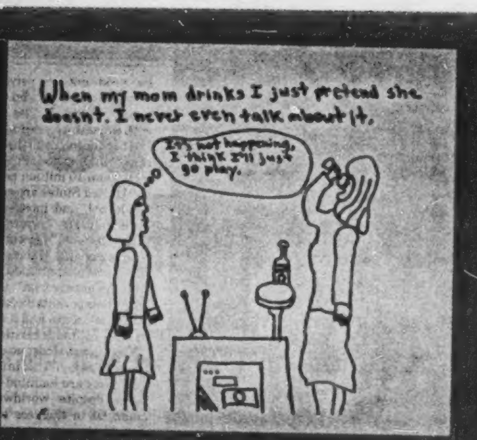
The anger of a COA cannot be seen by brain scans. But at a therapy session at Caron Family Services in Wernersville, Pa., Ken Gill, a 49-year-old IBM salesman, recently took a padded bat and walloped a couch cushion hard enough to wake up sleeping demons. "I came because I was hurting so bad and I didn't know why," he says. "A lot of things were going wrong. I

There's a Problem in the House

In "Adult Children of Alcoholics," Janet Geringer Woititz discusses 13 traits that most children from alcoholic households experience to some degree. These symptoms, she says, can pose lifelong problems.

Adult children of alcoholics . . .

- guess what normal behavior is.
- have difficulty following a project from beginning to end.
- lie when it would be just as easy to tell the truth.
- judge themselves without mercy.
- have difficulty having fun.
- take themselves very seriously.
- have difficulty with intimate relationships.
- overreact to changes over which they have no control.
- constantly seek approval and affirmation.
- feel that they are different from other people.
- are super-responsible or super-irresponsible.
- are extremely loyal, even in the face of evidence that the loyalty is undeserved.
- tend to lock themselves into a course of action without giving consideration to consequences.


When my mom drinks I just pretend she doesnt. I never even talk about it.
It's not happening. I think I'll just go play.

[illegible] year-old's nightmare—living in denial

was a workaholic, and I neglected my family." It took Gill only a few hours of exposure to the idea that he might be an "adult child," he says, to realize that his failings as a parent may be if not excused, then at least explained. Like a lot of kids who grew up in an alcoholic household, Gill, who is also a recovering alcoholic, never got what even rats and monkeys get: exposure, at an impressionable age, to the sight and sound of functioning parents. Suzanne Somers, the actress and singer, spent years working out her anger in the form of a just published book called "Keeping Secrets." "I decided that this disease took the first half of my life, and goddam it," she says, "it wasn't going to take the second half of it."

'Control freak': Not every COA has all of the 13 traits (chart, page 63) ascribed to them by Woititz in her landmark work, "Adult Children of Alcoholics" *(1983, Health Communications, Inc.)*, and not all have been scarred. (President Reagan, who has written of sometimes finding his father passed out drunk on the front porch, does not appear, from his famous management style, to suffer from any tendency to be a "control freak," a most common COA complaint.) Some children of alcoholics are grossly overweight from compulsive eating while others are as dressed for success as, well, Somers. A few COAs are immobilized by depression. Another runs TV's "Old Time Gospel Hour." What these people *do* have in common is a basic agreement with George Vaillant, a Dartmouth Medical School professor who says that it is important to think of alcoholism not as an illness that affects bodily organs but as "an illness that affects families. Perhaps the worst single feature of alcoholism," Vaillant adds, "is that it causes people to be unreasonably angry at the people that they most love."

The movement is only about six years old, but expanding so rapidly that figures, could they be gathered for such a basically unstructured and anonymous group, would be outdated as soon as they appeared. We do know, though, that five years ago there were 21 people in an organization called the National Association for Children of Alcoholics; today there are more than 7,000. The 14 Al-Anon-affiliated children-of-alcoholics groups meeting in the early '80s have increased to 1,100. With only word-of-mouth advertising, Woititz's book has sold about a million copies; indeed, "Adult Children of Alcoholics" reached the number-three spot on The New York Times paperback best-seller list long before it was available in any bookstore—at a time, in other words, when getting a copy meant collaring a clerk to put in an order and *saying the title out loud.*

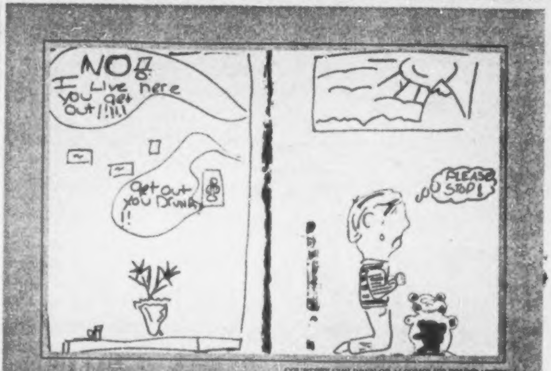
"We turned on the phones in 1982," says Migs Woodside, founder and president of the Children of Alcoholics Foundation in New York, "and the calls are still coming in 24 hours a day." The COAs Foundation sponsors a traveling art show that features the work of young and adult COAs; often, says Woodside, an attendee will stand mesmerized before a crude depiction of domestic violence or parental apathy ("Mom at noon," it says beneath the picture of someone huddling beneath the bedcovers)—and will then go directly to a pay phone to find help. "The newcomers all tend to say the same thing," says Woodside. '"Wait a minute—that's my story, that's *me!*"'

"It's private pain transformed into a public statement," says James Garbarino, president of the Erikson Institute for Advanced Study in Child Development, in Chicago, "a fascinating movement." But when you consider that denial is the primary symptom of alcoholism and that COAs tend by nature to take on more than their share of blame for whatever mess they happen to find themselves in, the rapid growth of the COAs movement seems just short of miraculous—something akin to a drunken stockbroker named Bill Wilson cofounding AA, now *the* model for a vast majority of self-help programs throughout the United States. After all, who would want to spill the family's darkest secret after years of telling teachers, employers and friends that everything was fine? ("A child of an alcoholic will always say 'Fine'," says Rokelle Lerner, a counselor who specializes in young COAs. "They get punished if they say otherwise.") Who would voluntarily identify themselves with a group whose female members, according to some reports, have an above-average number of gynecological problems, possibly due to stress—and whose men are prone to frequent surgery for problems, doctors say, that may be basically psychosomatic?

The answer is, only someone who had, in some sense, bottomed out, just the way a drinker does before he turns to AA.

The concept of codependency is at the center of the COAs movement. Eleanor Williams, who works with COAs at the Charter Peachford Hospital in Atlanta, defines codependency as "unconscious addiction to another person's dysfunctional behavior." Woititz, in a recent Changes magazine interview, referred to it more simply as a tendency to "put other people's

Talking and playing their way to a healthy state of mind:



COURTESY CHILDREN OF ALCOHOLICS FOUNDATION

■ The fighting never stops: Living with fear

needs before my own." A codependent family member may suspect that he has driven the alcoholic to drink (though that is impossible, according to virtually all experts in the field); he almost certainly thinks that he can cure or at least control the drinker's troublesome behavior. "I actually thought that I could make a difference by cooking my husband better meals and by taking the kids out for drives on weekends [so he could rest]," says Ella S., a Westchester, N.Y., woman. "For all I know, it's a deeply ingrained psychological, and possibly genetic, disease, and here I am going at it with a lamb chop."

Mental movies: Obsessed with her husband's increasingly self-destructive behavior, Ella's next step, in typical codependent fashion, was to hide Bob's six-packs, which made him, to put it mildly, angry. Soon they were fighting almost daily and Ella was running mental movies of their scenes from a marriage all night long. "I was wasting a lot of time and energy trying to change the past, while he kept getting worse," she says. "There was a kind of awkward violence between him and me all the time; our hearts weren't really in it, but it wasn't until he had an affair with an alcoholism counselor *that I got him to* that I left." If you're wondering about children, Ella has a seven-year-old daughter, Ann. Her omission is significant. If life were a horse race, then Ann has been, as they say on the past performance charts, "shuffled back" among the also-rans.

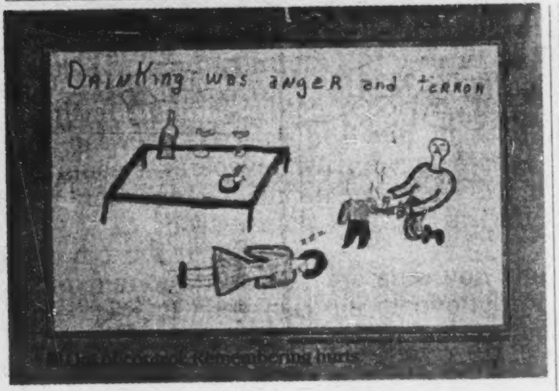
What COAs—all people affected by alcohol—need to learn is that the race is fixed: when there is no program of recovery—either through the support of a group or the self-imposed abstinence of an individual—the abused substance will always win, handily, no matter what the competition. The first step of AA begins, "We admitted we were powerless . . ." But what will become of Ann, who is codependent on *two* people? Perhaps, sensing that she is not exactly the center of attention, she will reach adulthood with a need for constant approval, a common COA symptom. Or maybe she will, even as a child, react to the chaos by trying to keep everything in her life under control, and thus give the impression that she is, despite everything, quite a trouper, a golden child.

"[Some] don't fall apart until they're in their 20s or 30s," says Woititz, and in some cases, especially those marked by violence or incest and sexual abuse (three times more common in alcoholic households than in the general population), that's the wonder of it all. One eight-year-old patient at Woititz's Verona, N.J., counseling center woke up in the middle of the night to see her alcoholic mother shoot herself in the head. "The child called the 911 emergency number, got her mother to the hospital and basically saved her mother's life," says Woititz. "When I saw her she was having

The board game Sobriety (left), Brooks with a father and son at her California counseling center

TOM IVES

RICK RICKMAN


DRINKING WAS ANGER AND TERROR

[illegible] Remembering hurts

nightmares—that she wouldn't wake up and witness this suicide attempt. This is not a normal nightmare. The child had become mother to her own mother."

Each unhappy family, as Tolstoy said, is unhappy in its own way. Artist Eric Fischl, 39, in a short videotape he made for the COAs Foundation called "Trying to Find Normal," speaks of stepping over his passed-out mother, in their comfortable-looking (from the outside) Port Washington, N.Y., home and seeing her "lying in her own piss." His work, which has been the subject of a one-man show at the Whitney Museum in New York, is not autobiographical, he says, and yet "the tone [of it] has everything to do with my childhood." His painting "Time for Bed" (page 63) "relates to my memory of all hell breaking loose," he says. "I guess you could say the boy is me and his shame, embarrassment and sadness is mine as well. The little boy's Superman pajamas are on backwards, so it's like looking in a mirror. I painted the woman standing on a glass table with spiked heels on to give it a sense of fragility and danger. The man only has one arm because I wanted a sense of impotence."

Alcohol leaves every alcoholic and codependent who does not admit his powerlessness over the substance in a constant state of longing. Fischl didn't realize how sad he'd been until his mother died, in an alcohol-related car accident, in 1970. "The thing about having a sick parent is that you think it's your problem," he says. "You feel like a failure because you can't save her." Even when there is no incest, there is seduction. Fischl's mother kept "signaling," he says, "that if you could just come a little bit further with me in this, you can save me."

Some of the other things that alcohol ruins, before it gets to the liver: family meals ("Alcohol fills you up. My father was never interested in eating with us"); gloriously run-of-the-mill evenings around the hearth ("Alcohol makes you tired. My father was in bed most nights at 8"). When enough C_2H_5HO is added to a home, vases may start to fly across the room and crash

Heredity and Drinking: How Strong Is the Link?

Research on the genetics of alcoholism took a curious turn a few weeks ago when Lawrence Lumeng analyzed his DNA to demonstrate why he can't tolerate liquor. Lumeng, a biochemist at the Indiana University School of Medicine, is among the 30 to 45 percent of Asians whose response to spirited beverages is a reddened face, headaches or nausea. This "Oriental flush," past studies have shown, arises in those who have an inefficient version of a liver enzyme that is crucial to the body's breakdown of alcohol; this "lazy" enzyme allows the buildup of an alcohol product, acetaldehyde, which is sickening and leads many Asians to shun alcohol. Working with biochemist Ting-Kai Li, Lumeng says that he pinpointed the gene that instructs cells to make the odd enzyme. The experiment offers dramatic evidence that a bodily response to alcohol is genetically dictated—and is thus inherited as surely as eye color.

There is no evidence for the opposite proposition: that a specific gene makes a person *crave* alcohol. Considering the wide variety of reasons why people consume the stuff, it seems unlikely that a "drinking gene" exists. But researchers have firmly established that, compared with other children, an alcoholic's offspring are around four times more likely to develop the problem, even if they were raised by other, nonalcoholic parents. In families with a history of alcoholism, explains C. Robert Cloninger, a psychiatrist and geneticist at Washington University in St. Louis, "what is inherited is not the fact that you are destined to become an alcoholic but varying degrees of susceptibility" to the disorder. So real is the predisposition that many researchers advise adult children of alcoholics (COAs) to drink no alcohol whatsoever.

Even the brains of COAs show faint signs of unusual activity, according to controversial studies by psychiatrist Henri Begleiter of the State University of New York in Brooklyn. Begleiter has found that young boys who have never consumed alcohol produce the slightly distorted brain-wave patterns typical of their alcoholic fathers. Such signature brain waves, he says, may mark the son of an alcoholic as likely to develop a drinking problem and perhaps alert him to the risk. However, it

MARY ANN CARTER

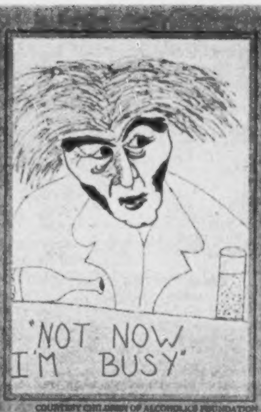
Probing for genes: *Lumeng*

into walls. All kinds of paper—court-issued Orders of Protection, divorce decrees, bounced checks—come fluttering down. The lights go on and off. Does that mean Daddy's forgotten to pay the bill again, or that the second act is starting?

Every alcoholic household is, in fact, a pathetic little play in which each of the members takes on a role. This is not an idea that arrived with the COAs movement; a 17-page booklet called "Alcoholism: A Merry-Go-Round Named Denial" has been distributed free of charge by Al-Anon for almost 20 years. Written by the Rev. Joseph L. Kellerman, the former director of the Charlotte, N.C., Council on Alcoholism, "Merry-Go-Round" takes note of the uncanny consistency with which certain characters appear in alcoholic situations. These include the Enabler ("a 'helpful' Mr. Clean . . . [who] conditions [the drinker] to believe there will always be a protector who will come to his rescue"); the Victim ("the person who is responsible for getting the work done if the alcoholic is absent") and the Provoker (usually the spouse or parent of the alcoholic, this is "the key person . . . who is hurt and upset by repeated drinking episodes, but she holds the family together . . In turn, she feeds back into the marriage her bitterness, resentment, fear and hurt . . . She controls, she tries to force the changes she wants; she sacrifices, adjusts, never gives up, never gives in, but never forgets").

Some of the earliest books in the COAs movement explored the drama metaphor more deeply and defined the roles that children play. Sharon Wegscheider-Cruse, in her 1981 book, "Another Chance" *(Science and Behavior Books, Inc. Palo Alto, Calif.)*, wrote about the Family Hero, who is usually the firstborn. A high achiever in school, the Hero always does what's right, often discounting himself by putting others first. The Lost Child, meanwhile, is withdrawn, a loner on his way to a joyless adulthood, and thus, in some ways, very different from the Scapegoat, who appears hostile and defiant but inside feels hurt and angry. (It is the Scapegoat, says Wegscheider-Cruse, who gets attention through "negative behavior" and is likely to be involved in alcohol or other drugs later.) Last and least—in his own mind—is the Mascot, fragile and immature yet charming: the family clown.

'Good-looking' kids: Virtually no one was publishing those kinds of thoughts when Claudia Black, a Laguna Beach, Calif., therapist, began searching for literature on the subject of the alcohol-affected family in the late '70s. "Half of my adult [alcoholic] patients had kids my age and older," she remembers, "but all I found was stuff on fetal alcohol syndrome and kids prone to juvenile delinquency." One thing that fascinated her about young COAs, she says, was that despite their developmental problems "they were all 'good-looking' kids"—presentable and responsible albeit



COURTESY CHILDREN OF ALCOHOLICS FOUNDATION

■ Trauma: Parental neglect

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remains to be seen whether such brain scans are sufficiently reliable and informative to distinguish potential social drinkers from future alcoholics. The technique, comments psychologist Robert Pandina, scientific director of the Center of Alcohol Studies at Rutgers University, is "at this time not any more valuable" as a predictor of future drinking behavior "than collecting a good family history on an individual."

Other studies show that many COAs respond uniquely to booze. Marc Schuckit, a psychiatrist at the Veterans Administration Hospital in San Diego, has found that college-age sons of alcoholics often react less to a few drinks than other college men; in his studies, the drinkers' sons were generally not as euphoric or tipsy after three to five cocktails. Schuckit believes that this lower sensitivity makes it harder for the alcoholics' sons to know when to stop drinking, starting them down the road to alcohol problems. Preliminary experiments by Barbara Lex of McLean Hospital in Belmont, Mass., confirm that daughters of alcoholics respond similarly. Women from families with a history of alcohol abuse tend to keep their balance better on a wobbly platform after having a drink. Apparently women, too, can inherit traits that might predispose them to addiction, although there are far fewer female than male alcoholics.

Half a beer: The key unresolved issue, of course, is why some individuals from alcohol-scarred families succumb to alcoholism while others don't. Genes play some role in the development, most notably in abstinence. "People say that whether you drink or not has to do only with willpower," explains Indiana's Lumeng, "but the reason I can drink only half a beer is biological."

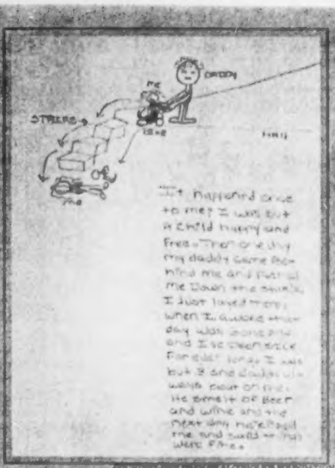
Yet heredity alone obviously isn't to blame for alcoholism's appalling toll. In fact, about 60 percent of the nation's alcohol abusers are from families with *no* history of the disorder. How much people drink is influenced by factors as prosaic as cost; partly to curb consumption, the National Council on Alcoholism is lobbying to raise federal excise taxes on beer and wine, which haven't changed since 1951. Social influences like cost and peer pressure "are just as important as genes," says Dartmouth psychiatrist George Vaillant. "All the genes do is make it easier for you to become an alcoholic." For now, the value of genetic studies is to warn COAs that they may well have a real handicap in the struggle against the family trouble.

TERENCE MONMANEY *with*
KAREN SPRINGEN *in New York*
and MARY HAGER *in Washington*

Tipsy? *Lab demonstration*

not terribly verbal. "They had friends but weren't honest with them. Everything was 'fine and dandy'."

The title of Black's important 1981 book, "It Will Never Happen to Me" *(M.A.C. Denver, Colo.)*, reflects the typical codependent's mix of denial and false bravado. In it, she makes the point that the children in an alcoholic household never have an environment that is consistent and structured, two of the things they need most—and she, too, talks of such stock juvenile "roles" as the Responsible One and the Adjuster. Her unique warning was that children who survive a parent's alcoholism by displaying unusual coping behavior often experience "emotional and psychological deficits" later on. They are also likely to become alcoholics, says Black, because "alcohol helps these persons become less rigid, loosen up and relax. When they drink, they aren't quite so serious." Though those things happen to almost everyone who imbibes, Black says that "for those who are stuck in unhealthy patterns, alcohol may be the *only* thing that can provide relief."



COURTESY CHILDREN OF ALCOHOLICS FOUNDATION

■ Physical abuse: An adult remembers

Well, she guessed wrong there: a movement, manifested by often joyous meetings, has come along in the interim. At hundreds of COAs gatherings around the country tonight, people will talk and listen to each other's stories, to cry, to laugh and generally, as Ken Gill says, "recharge their batteries." "This program kept me from being an alcoholic myself," said a woman named Heather at a gathering in an affluent section of San Francisco last week. "Because I was the oldest, everything was always my fault. It's like when you make your parents breakfast and you bring them one scrambled egg and one fried egg—in my house I always scrambled the wrong egg." Heads bobbed in agreement. Who else but COAs could identify with a story about what happens when kids cook for their own mother and father?

Discovering self-esteem: Talking and listening: this is the way we've learned to deal with problem drinking. And though it sounds wimpy, don't knock it; it's the surest way to alleviate not just the imbibing but the whole range of symptoms we call alcoholism. A woman named Nina stood up at a meeting in Boston last week, practically glossed over the fact that both her parents were alcoholics—and proceeded to speak about how well she was feeling and doing. COAs meetings and literature, she said, had allowed her to discover self-esteem. At another meeting, Carolyn told a story of complaining to her doctor about depression—and hearing the doctor shoot back a question about whether one of her parents was an alcoholic. "I was shocked," she said, and well she might be. Doctors, as a group, have yet to play a major role in helping mitigate the effects of alcohol, perhaps because the average medical-school student spends a grand total of between zero and 10 hours studying the affliction that kills 100,000 people annually.

An avalanche of information is coming, nevertheless, from another kind of M.D.—call them the Masters of Disaster, the people who've lived with alcoholism or worked with alcoholics so closely that they might as well be their kin. Robert Ackerman, a professor of sociology at Indiana University of Pennsylvania, has been studying the children of alcoholics for an exceedingly long time by the standards of the movement—since the early '70s. In his recent book "Let Go and Grow" *(Health Communications, Inc.)*, he reports on a survey he took to test the validity of Woititz's 13 generalizations about COAs, as well as seven more observations of his own. What he found was that "adult children of alcoholics identified about 20 percent more with these characteristics" than did the general population. Other professionals are reporting success with therapies involving hugging, acting out unresolved scenes from long ago and even playing one of several board games for children of alcoholics called Family Happenings and Sobriety. Cathleen Brooks, executive director of a program called Next Step in San Diego, reports that her clients often make life-changing strides after six to 18 months of primary treatment and make the decision never to drink or take drugs.

A founding mother of the movement: *Woititz*

BERNARD GOTFRYD—NEWSWEEK

The 7 million COAs who are under the age of 18 are harder to help, if only because their parents' denial tends to keep them out of treatment. For these children who never know what to expect when they come home from school each day, life, says Woititz, "is a state of constant anxiety." Some pediatricians think there is a link between such anxiety and childhood ulcers, chronic nausea, sleeping problems, eating disorders and dermatitis. Migs Woodside, from the COAs Foundation, says that the trained teacher can pick the child of an alcoholic out of a crowded classroom. "Sometimes you can tell by the way they are dressed or by the fact that they never have their lunch money," she says. "Sometimes you can tell by the way they suddenly pay attention when the teacher talks about drinking, and sometimes you can tell by their pictures."

Someday, 20 or 30 years from now, those children may feel a vague sense of failure or depression and be hard pressed to explain why. In the meantime, it's their Crayolas that are hard pressed. Beer cans—and not liquor or wine bottles—form a leitmotif in the work of young children of alcoholics. Occasionally, Woodside says, looking a little sad, the big stick figures can be seen tipping the cans into the mouths of the little stick figures.

CHARLES LEERHSEN *with* TESSA NAMUTH
and bureau reports

COMING TO GRIPS WITH ALCOHOLISM

■ James McKelvey looked up last week from a plate of beef and potatoes, took a swig of Diet Coke and shook his head in dismay at what the government's lawyers were telling the Supreme Court about him—that he was an alcoholic because he was short on will power. "My doctor told me that if I drank again, I would die. And I tried and tried with all the power I had to quit. I told my wife a hundred times I would never drink again. I didn't say it to pacify her. I honest to God meant it. But then I'd turn around 180 degrees and want a drink even more."

These days, McKelvey is sober. In fact, he hasn't had a drink since 1975, since the day he killed off a fifth of bourbon and had to be persuaded by a recovered alcoholic priest not to kill himself. Now, he and another alcoholic Army veteran, Eugene Traynor, are battling not the bottle but the Veterans Administration, for refusing to recognize their alcoholism as a disability that would qualify them for special treatment when it comes to applying for college benefits—they missed the application deadline because they were drunk for years. Their lawsuits, set for a December 7 hearing, could result in a landmark ruling. The question before the Supreme Court: Is most alcoholism simply a result of "willful misconduct," as the VA contends, or is it a disease in which the victim is compelled to drink?

At stake for society

Not only does the case jeopardize the premise upon which virtually every program in America's $1 billion alcoholism-treatment industry is based. It also gets to the heart of how society views people in the grip of alcohol. Among the perplexing questions that some see arising: If alcoholism isn't a disease, will employers quit paying treatment costs for alcoholic workers and start firing them? If it is a disease, does it provide an appropriate defense against wrongdoing? Already, some critics of the disease concept are pointing a finger at Michael Deaver, the former White House aide on trial for perjury, who now is telling the world that he's an alcoholic. If Deaver can successfully argue that he should be excused for lying because he was sick, critics worry that alcoholism will be used to condone all sorts of illegal acts. "Where does it all end?" asks one scholar.

The issue of responsibility is coming

■ Nearly 18 million adults in the U.S. are problem drinkers, according to the government's latest report on alcoholism and alcohol abuse. More than 10 million of these drinkers are suffering from alcoholism.

■ Alcohol is a factor in nearly half of America's murders, suicides and accidental deaths. In all, it claims at least 100,000 lives per year, 25 times as many as all illegal drugs combined.

■ Two of every 3 adults drink. But only 10 percent of the nation's drinkers consume half of its beer, wine and liquor. The South and the West have the most abstainers. But their drinkers drink the most.

■ Two of every 3 high-school seniors have drunk alcohol within the past month. Five percent drink daily. Forty percent of sixth-graders have tasted wine coolers. By age 18, a child will have seen 100,000 beer commercials.

'THE SICK WANTING TO BE WELL'

"I control you. You will always want me. You will always need me," a teenager posing as "Alcohol" tells a gym full of San Francisco students. He and other recovering alcoholics are in Teens Kick Off, a troupe with this message: "Chemical-dependent people aren't bad wanting to be good but sick wanting to be well"

up just as safety officials are starting to notice an alarming trend on the highways. Traffic fatalities, which had steadily declined in this decade, spurted up 5 percent last year, and a surge in drunken driving among young people seems partly to blame. Ralph Hingson of the Boston University School of Public Health analyzed data from 15 states and found, for the first time since 1980, an increase in fatal one-car crashes at night—accidents that usually involve alcohol. This 7 percent rise occurred, Hingson noted, after a peak in the national crusade against drunk driving. "The drunk-driving issue has lost some of its steam. We ought to be concerned."

The disease dispute also is taking hold in a year in which studies emerge almost monthly suggesting that a susceptibility to alcoholism may be inherited. One example: Boys who don't drink but who have alcoholic fathers tend to have the same brain-wave patterns.

Whatever the cause, excessive drinking is taking a heavy toll on American life. A Gallup Poll this year showed 1 in 4 families troubled by alcohol—the highest incidence of problem drinking in 37 years. The government estimates that 10.6 million adults—a record number—are alcoholics. Seven million more are problem drinkers, many of them young men who drink only episodically but still get drunk enough to wind up in fistfights or car wrecks. (Ironically, per capita consumption of alcohol has dropped slightly in this decade, in part because men age 18 to 25—the heaviest drinking group—are making up a smaller share of the population.) Last year, when basketball star Len Bias died from a cocaine overdose, the country was swept up in an antidrug fervor. But in the same year, alcohol took more than 100,000 lives—25 times as many as claimed by cocaine, heroin and other illegal drugs combined. That's the government's estimate, based largely on death certificates. One recent study indicated that many death certificates cover up links to alcohol.

Flushed Asians, Drunk Monks

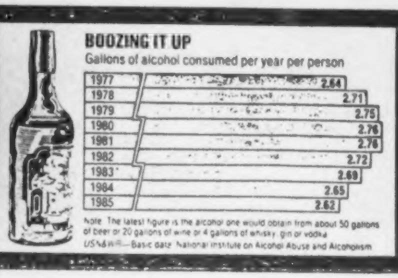
No ethnic group is spared, though some are hit harder than others. Alcoholism is like a wildfire, says National Black Alcoholism Council Chairman Maxine Womble, but "in the black community the fire tends to spread much,

- The economic costs to society of alcoholism and alcohol abuse are estimated at nearly $117 billion a year—including $18 billion from premature [illegible]ths, $66 billion in [illegible]ced work effort, $13 billion for treatments.

- One family in 4 has been troubled by alcohol—the highest incidence of problem drinking in a Gallup trend that dates back to 1950. But Gallup finds only 17 percent of Americans in favor of a return to Prohibition.

BOOZING IT UP

Gallons of alcohol consumed per year per person



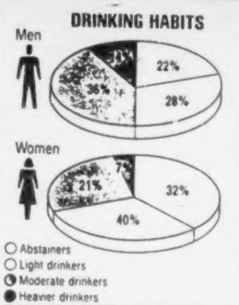
Note: The latest figure is the alcohol one would obtain from about 50 gallons of beer or 20 gallons of wine or 4 gallons of whisky, gin or vodka

USN&WR—Basic data: National Institute on Alcohol Abuse and Alcoholism much faster and burn a whole lot longer." It remains a common problem for American Indians and people of Irish descent. It's also showing up more among groups in which it was always rare—among Jews, who traditionally frowned on intoxication, and among Asians, even though many Orientals feel uncomfortable and develop a skin flush on drinking alcohol. All occupations are vulnerable. In Atlanta, attorneys belong to an Alcoholics Anonymous chapter called "Legal Eagles." Pilots in the city are in "Birds of a Feather." Ministers get together as the "Drunk Monks."

New Year's binge

Even healers are victims, as Dr. Lyman Boynton of Berkeley, Calif., can personally report. "You can't be an alcoholic," he used to tell himself time and again. "You're a surgeon." But telltale signs kept giving him away. He arranged his on-call schedule so he could drink undisturbed. He promised his children—over and over—that he would quit. His last pledge came five Decembers ago. He would stop drinking by January 3. "But I woke up on the third with the shakes. I was scared. That's when I called for help."

What's especially troubling to people who try to treat alcoholism is its changing face. Once considered mainly a male condition, it now affects large numbers of women. It probably always did, some experts say. Until recently, men were viewed as suffering from alcoholism while women were diagnosed as victims of depression, panic attacks and anxiety. Today, more doctors are asking troubled women about their drinking patterns and concluding that alcohol may be the culprit. Women, once rare in AA, now make up one third of its membership.



These figures are for persons with full-time jobs. Among part-time workers, 35% of men and 4% of women are heavy drinkers.

Note: Abstainers consume 0-12 drinks per year, light drinkers have 3 drinks per week, moderate drinkers have 4-13 drinks per week and heavier drinkers have 14 or more drinks per week.

USN&WR—Basic data: National Institute on Alcohol Abuse and Alcoholism

Alcoholism also is swapping its image as a middle-aged woe for a more youthful look. "You can see a whole AA meeting of people without gray hair," says Hannah, 51, an AA member in Los Angeles. "I'm the senior citizen in our group. People are hitting rock bottom and seeking help at 25, a lot earlier than they used to."

Whisky at 11, tequila at 12

Some hit bottom even sooner. Kent T. was a 10-year-old in San Marino, Calif., when he began drinking his parents' beer. He was hooked on whisky at 11. At 12, he started his days with tequila. Three years ago, at 14, he had one beer at a Deep Purple rock concert and blacked out. "I woke up feeling like a .22 bullet was ricocheting around inside me." That's when he got help.

Even though every state but Wyoming has raised its minimum drinking age to 21, many social workers detect little if any slowdown in drinking among teenagers and even preteens. In fact, some parents are relieved that their children are drinking instead of using drugs. An introductory drink is often a fruit-flavored wine cooler, which many children consider soda pop. A recent *Weekly Reader* survey indicated that 2 of every 5 children have had wine coolers before their 13th birthday. Among teenagers, beer remains No. 1. A possible factor: By age 18, American children on average see an estimated 100,000 beer commercials.

Many young drinkers are groping with multiple addictions—cocaine, heroin, even eating disorders, plus alcohol. "The days of the garden-variety alcoholic are over," says Carl Sissom, adult-program director at Parkside Lodge in suburban Chicago. "Most are poly drug users." The trend became so strong that the National Association of Alcoholism Treatment Programs recently changed its name to the National Association of Addiction Treatment Providers. "Even a coke head has to sleep," says NAATP President Michael Ford. "When he does, he usually turns to alcohol or tranquilizers." And alcoholics resort to drugs to feel different or treat hangovers. This is seen often by Dr. Anne Marie Riether, director of Emory University's Addictive Disorder Program in Atlanta. "It's like switching seats on the *Titanic*."

The multiple addictions of

A FAMILY WITH A HISTORY

"I'm a surgeon," Lyman Boynton of Berkeley used to tell himself. "I can't be an alcoholic." But he is. So are two of his children, Chris, 20, left, and Ursula, 17, second from right. The doctor has been sober six years, the children four. Kirsten, 22, and Benjamin, 16 and adopted, are not alcoholics

Billy T., 28, of Los Angeles were bizarre but not uncommon. "My first ubstance of abuse was food, especially sugar," he recalls. At 15, he started drinking his parents' vodka. At 16, he developed a marijuana habit. As a 250-pound college student, he searched garbage cans along fraternity row for uneaten pizzas, which he warmed with a hair dryer. Grain alcohol became his drug of choice. A friend found him out of work and suicidal and persuaded him to go to Overeaters Anonymous, then AA. Now he's sober and down to three meals a day. "It's a disease, a cunning, sneaky disease," he says. "It waits for you like the Grim Reaper."

The fearful respect that Billy T. voices for alcoholism is ordinary fare among America's 676,000 AA members. If alcoholism is anything, they believe, it's permanent. An alcoholic is never cured, AA contends, and the only way one can stay sober is to not drink at all: "One drink, one drunk." This clear-cut view has helped AA sober up more alcoholics than has any other treatment program. Even professionals who work with alcoholics outside AA rely on it heavily.

The idea that alcoholism is a sickness isn't new. Benjamin Rush, a Philadelphia physician who signed the Declaration of Independence, concluded that alcohol was a drug and habitual drunkenness involuntary. He saw only one cure: Total abstinence. But the view that alcoholics weren't sick but sinful persisted in American society until well into this century. In 1957, a landmark resolution by the American Medical Association declared alcoholism a disease. Later came this definition from the AMA: "Alcoholism is an illness characterized by preoccupation with alcohol and loss of control over its consumption such as to lead usually to intoxication if drinking is begun, by chronicity, by progression, and by tendency toward relapse."

The public is satisfied with that definition. A recent Gallup Poll found 87 percent of those interviewed endorsing the disease concept. The gradual shift in opinion has taken much of the stigma away from alcoholism and made the alcoholic a person to be helped and treated rather than scorned and jailed.

Should nonalcoholics worry?

There's a flip side. "The disease concept is a wonderful cop-out for society," says Robin Room, director of the Alcohol Research Group at the Medical Research Institute of San Francisco. "It says that there's normal drinking and there's alcoholic drinking and if you're not an alcoholic you don't have to worry." The disease concept, argues Room, "was the compromise that the alcoholism movement in the 1940s and '50s had to make to get any attention. They were saying, 'Look, we're not the temperance movement, and we're not trying to dash the cup from your lips. We're just trying to get help for alcoholics.'"

One scholar who thinks this effort is misguided is Herbert Fingarette, an expert on addiction at the University of California at Santa Barbara and author of an upcoming book, *Heavy Drinking: The Myth of Alcoholism as a Disease*. The government's legal brief in the VA case often turns to Fingarette to back the view that alcoholism is a behavior problem. Scientific studies have yet to show, he says, that alcoholics cannot willfully quit drinking. "We have cultural beliefs about alcohol, and one is that if you drink, you become less responsible for what you are doing," Fingarette says. "Alcoholism is a label and it shouldn't be a blanket excuse for anything."

Alcoholic veterans Traynor and McKelvey have a date with the Supreme Court

Many specialists worry that the flow of money into treatment programs—from employers, insurance companies and governments—may ebb if the Supreme Court undermines alcoholism's status as a disease. "A billion-dollar industry services alcoholism as a disease," says San Francisco psychologist Paul Good. "If you call it a moral problem, you don't have a treatment industry." Some lawyers doubt that the Court will go that far. It had a chance to do so in a criminal case challenging a Texas law against public drunkenness 19 years ago and demurred. In doing so, the Court noted that there were differences of opinion among medical experts on whether alcoholism is a disease.

If the debate goes on, it will be a battle of politics and semantics as much as science and medicine. What interests many researchers much more is what's happening in genetics. Dozens of studies over the years showed alcoholism run-

SMALL MOLECULE, LARGE EFFECTS

The devastation that alcohol wreaks on the liver and brain of chronic, heavy drinkers is legendary. But alcohol begins to alter the functioning of virtually every organ from the moment it enters the body. Because it is chemically a small molecule, alcohol is readily absorbed by the walls of the gut into the bloodstream, where it is transported throughout the body. Alcohol can also pass through the placenta to the developing fetus and be transmitted through breast milk to newborns.

The bulk of medical evidence indicates that drinking in moderation is not harmful to adults. Some controversial studies even suggest that a drink or two a day extends the life span by preventing coronary-artery disease. But the dangers of consuming large quantities of alcohol seem to mount with every new study. At high blood levels, alcohol disrupts cell membranes, impairing the flow of nutrients and other critical biochemicals into and out of the cell.

Common myths

Contrary to popular belief, alcohol is a depressant or tranquilizer, not a stimulant. And the apparent warming effects of a drink are an illusion: Alcohol causes the blood vessels to dilate, increasing the flow of blood to the skin. While that may make you feel warmer, in reality the shift of blood flow to the body's surface can speed the loss of body heat in cold weather.

The time it takes for alcohol concentration to peak in the blood depends on a number of factors, including how much food is in the stomach prior to drinking. It takes the liver about an hour to break down the alcohol in a single drink (1½ ounces of liquor, a beer or a glass of wine) into less harmful products for excretion. But mental impairment may persist long after the liver has finished its job. In a study in Sweden, subjects were given a driving test the morning after a night of binging; even those who felt fully sober and who had no alcohol left in their blood showed a marked decline in performance.

Black coffee and other time-honored remedies do nothing to speed up the brain's recovery. Although the stimulant properties of caffeine may slightly counteract the tranquilizing effects of alcohol, the impaired reaction time that alcohol causes is unaltered.

Chronic drinking often leads to tolerance. The individual must imbibe more and more alcohol to get the same high. Once consumption exceeds more than three to four drinks a day, the dangers rapidly outnumber the benefits. Alcoholic beverages contain calories, but negligible nutrients. And as Shakespeare aptly described the effect of alcohol on sex: "It provokes the desire, but it takes away the performance."

Damage to the body can begin to occur after only a few weeks of heavy drinking. As the liver becomes overtaxed, it loses its ability to break down alcohol and other toxins.

Perhaps the most tragic consequence of alcohol abuse is the mental retardation and birth defects that occur in one third of children whose mothers had six or more drinks a day during pregnancy. Three drinks a day lowers the risk to 10 percent. But some experts believe even occasional drinks could be dangerous if the fetus is exposed during a critical phase in brain development. As the U.S. surgeon general warns, no one knows what level of alcohol is safe during pregnancy.

by Kathleen McAuliffe

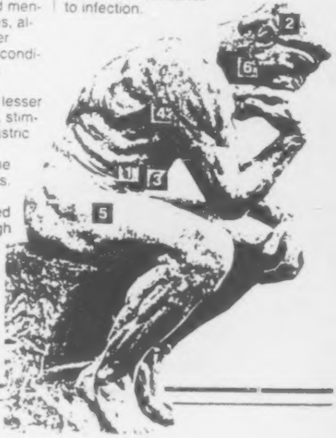
WHERE ALCOHOL TAKES ITS TOLL ON THE BODY

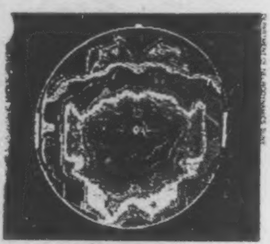
1 LIVER: About 1 in 3 heavy drinkers will develop scars on the liver associated with cirrhosis—a disease in which liver cells are destroyed and the organ no longer is able to process nutrients in food. The disease is usually painless until the damage is too far advanced to be treated. About half of those who develop cirrhosis will die within five years. Although the disease is generally associated with very heavy drinking—the average patient in one survey had consumed 13 drinks a day for 20 years—a recent French study suggests that men who have as little as three drinks a day, and women who take 1½ drinks a day, may be at increased risk of developing cirrhosis.

2 BRAIN: Heavy abuse of alcohol causes a loss of cells in different regions of the brain and also disrupts connections between nerve cells. That leads to permanent damage of the brain and nervous system. Among such chronic effects are polyneuropathy (the loss of sensation) and Wernicke's disease, typified by paralysis of eye movement and rapid mental deterioration. In severe cases, alcohol withdrawal can also trigger hallucinations and tremors, the condition known as delirium tremens.

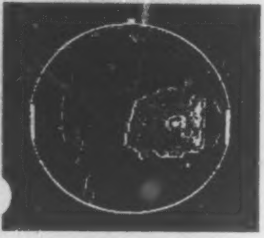
3 STOMACH: Wine, and to a lesser degree beer and diluted whisky, stimulate the stomach to secrete gastric acid. The lining of the stomach becomes inflamed, leading to the formation of painful peptic ulcers. Heavy drinkers also frequently suffer from diarrhea and impaired ability to absorb nutrients through the intestines.

4 HEART: Chronic abuse of alcohol frequently causes hypertension, or high blood pressure, increasing the risk of strokes and heart attacks. It can also damage the muscles of the heart directly, causing a condition known as cardiomyopathy. In addition, alcohol inhibits the manufacture of red and white blood cells, which may result in anemia and weakened resistance to infection.





Brain damage: The dark blue area in the scan below shows depressed electrical activity for an alcoholic. A normal brain scan, above, shows no such problem



5 REPRODUCTIVE SYSTEM: A significant rise in sex hormones occurs shortly after alcohol enters the blood system. But at high doses, the level of the hormones falls. With heavy alcohol use, both sexes may fail to reach a climax, and men may lose the ability to sustain an erection. Alcohol is in fact a major cause of male impotence. And drinking by women during pregnancy carries the serious risk of harming the fetus.

6 CANCER: Long-term alcohol abuse causes a striking increase of cancers of the digestive tract. The mouth and throat are most vulnerable—especially if the drinker also smokes tobacco. But cancers of the liver, stomach and colon are also much more common in alcoholics than in the general population. Women may run the additional risk of developing breast cancer. Although the evidence is still not conclusive, one recent study found that taking just three to nine drinks a week may increase the chance of breast cancer by 30 percent.

ning in families, with young people driven to drink by the stresses of life in a home ruled by drink. Now, there's growing evidence that some children of alcoholics inherit certain physiological traits that may indicate a tendency toward alcoholism. "We can't take someone who hasn't started boozing yet and predict if he's going to be a drinker or not," says Dr. Donald Goodwin, chairman of the psychiatry department at the University of Kansas Medical Center. But that's a major goal.

An early piece in the puzzle was a large study of adopted children in Sweden. It showed that about a third of the biological sons of alcoholic fathers became alcoholics themselves—three times the frequency among sons of nonalcoholics. Dr. Robert Cloninger, a professor of psychiatry and genetics at Washington University, found the influence of heredity most pronounced in males who became alcoholics in their teens. In addition, both fathers and sons seemed to experience the same troubles—blackouts, violent outbursts, drunken-driving arrests.

Other studies seem to support the genetic hypothesis. Dr. Marc Schuckit of the San Diego Veterans Hospital tested college students who were the sons of alcoholics but who were not alcoholics themselves. Compared with the sons of nonalcoholics, they felt less drunk with the same level of alcohol circulating in their blood. They also swayed less, slurred their words less and had better hand-eye coordination. A recent study of college-age women in Boston showed similar results. The heightened tolerance for alcohol, says Dr. Jack Mendelson, professor of psychiatry at Harvard Medical School, may make the children of alcoholics likely to drink larger doses of alcohol. "It doesn't take much imagination to see how they become trapped in a vicious cycle of dependency."

Whether the cause is genetic or environmental—and most experts think it's a combination of both—millions of children from alcoholic homes turn out to be alcoholics themselves. Sons of alcoholic fathers are four times more likely to become alcoholic than sons of nonalcoholics. Daughters of alcoholic mothers are thrice as likely to have the problem.

The children of alcoholics—known now as COA's—once were ignored by social workers. The assumption was that the alcoholic was the problem, and once the drinker was healed, so was the family. Today, the idea of co-dependency—that alcoholism negatively affects whole families—is gaining ground and fostering an array of support groups.

At present, 7 million children below the age of 18 live in alcoholic homes. Specialists who work with COA's say most suffer feelings of guilt and self-hate and usually live by three rules: "Don't talk. Don't trust. Don't feel."

An additional 21 million Americans are known as ACOA's, "adult children of alcoholics." Some do well. Many don't. "ACOA's are people with adult bodies and intellects and the wounded feelings of a child," says Sharon Wegscheider-Cruse, who runs a treatment program at the Onsite Center in Rapid City, S.D. For a $1,200 fee, often covered by insurance, the center puts ACOA's through eight days of therapy and role-playing aimed at "helping them go back and capture that feeling part of themselves that got lost in childhood."

The challenge of turning around hard-core alcoholics is much tougher, but nearly 1,000 treatment centers plus 33,840 AA groups are giving it their best shot. Most AA groups take in barely $1 or $2 per member per meeting—donations to buy doughnuts and coffee—but for many of the others, treatment is big business. "This is an industry, an important growth sector of the economy," says NAATP's Ford.

Inpatient vs. outpatient

One problem with this growth is that no one has found conclusive answers on which type of treatment is best for which type of alcoholic. A growing number of insurance plans favor inpatient treatment in a hospital, which can cost up to $15,000 for a 28-day stay. Residential units offer virtually the same services but not in a hospital setting; they often cost less than $10,000. Some experts believe that outpatient programs involving 4 hours of therapy and education each evening work just as well. They let patients stay at their jobs and usually cost $3,000 or less. But insurance companies, which base their fees on the size of their claims, usually pay minimal amounts for outpatient treatment while reimbursing generously for hospital care.

All programs start with a weaning of the alcoholic from alcohol. Most hospital centers use sedatives to help patients get through withdrawal symptoms, which persist for days or weeks. A few even employ acupuncture. Once sober, patients undergo extensive counseling and group-therapy sessions.

The U.S. is one of the few nations in which the treatment goal is almost always total abstinence. Many foreign specialists, especially in Britain and the Scandinavian countries, strive to sober up heavy drinkers and then show at least some—but not all—how to drink in moderation. William Miller, a University of New Mexico psychologist who believes in the concept, ran an ad offering to help people reduce their drinking and got very few calls. "Contrast this to an ad placed by an Oslo psychologist," says Miller. "She got hundreds of phone calls, so many they couldn't handle the demand."

AA vigorously opposes controlled drinking. Anyone who can stop at a few drinks, it contends, isn't really an alcoholic. Most professionals in the U.S. also are wary of controlled drinking. Says Thomas Seessell, executive director of the National Council on Alcoholism: "We'd be playing with fire. Our knowledge is not at a point where we can identify in advance who the candidates should be."

Acupuncture goals: Ease withdrawal symptoms, reduce craving

That, in fact, is the toughest challenge in nearly every case of alcoholism—matching the patient with the proper treatment. George Jacobson, director of research and clinical training at Milwaukee's DePaul Hospital, urges his students to think about cancer when they think about alcoholism. "The last time I looked, there were 82 different types of cancer and they all required different kinds of treatment." Alcoholism, too, may have different forms, Jacobson says, and need many different treatments.

Do any treatment programs really work? AA claims that at least half the people who stick with its nonmedical, self-help program stay sober. But outside researchers—AA is reluctant to let them inside—believe that perhaps 4 out of 5 people who go to AA meetings soon drop out. Scientific journals estimate that no treatment effort has a success rate over 35 percent. A lot depends on the patient. "The longer one is an alcohol abuser, the harder it is to change that behavior," says Dr. George Vaillant, professor of psychiatry at Dartmouth Medical School.

Until much more is accomplished, says psychologist Stanton Peele, "the greatest cure for drinking is just getting older." According to a number of studies, most people who abuse alcohol or drugs in their teens and early 20s rein in their excesses by their early 30s. "It's not that treatments don't work," says Peele, author of *The Meaning of Addiction*. "It's that they don't do much more than the natural growth process."

But don't tell that to Lyman Boynton, the doctor who went on the wagon five Januaries ago. He's certain that much can be done. In fact, his own experience has been so positive that he has quit his job as a surgeon in order to run a substance-abuse program at San Francisco's Kaiser Permanente Hospital. Now, he says, "I have all the things in recovery that I was looking for in a bottle." ■

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by Lewis J. Lord with Erica E. Goode, Ted Gest, Kathleen McAuliffe, Lisa J. Moore, Robert F. Black, Nancy Linnon and bureau reports

GUIDE TO GETTING HELP

TO FIND A TREATMENT PROGRAM, CONSULT—

■ Your family doctor

■ Alcoholics Anonymous
P.O. Box 459
Grand Central Station
New York, N.Y. 10163
(212) 686-1100 or call a local AA office

■ National Clearinghouse for Alcohol and Drug Information
P.O. Box 2345
Rockville, Md. 20852
(301) 468-2600
Extensive list of treatment programs as well as information on specific alcohol and drug problems

■ National Council on Alcoholism
12 West 21st Street
New York, N.Y. 10010
(800) NCA-CALL

■ *Roads to Recovery*, edited by Jean Moore (Macmillan, New York, 1986), lists and describes briefly several hundred treatment facilities nationwide

THEN—

Ask about the philosophy of the program. Make sure it's something you can live with, that it makes sense to you. Ask how much family members are involved in the program. Find out what happens if something goes wrong during treatment and what sort of follow-up is provided after treatment. And ask about licensing and accreditation. There are two accreditation boards: Commission on Accreditation of Rehabilitation Facilities and the Joint Commission on Accreditation of Health Care Organizations

FOR FAMILIES OF ALCOHOLICS:

■ Al-Anon Family Group Headquarters, Inc.
P.O. Box 862
Midtown Station
New York, N.Y. 10018-0862
24-hour answering service:
(800) 356-9996 (including Alaska, Hawaii, Puerto Rico and the Virgin Islands)
(212) 245-3151 (New York and Canada)
Referrals to local-group meetings that provide help for families of alcoholics

■ Adult Children of Alcoholics
Suite 200
2522 West Sepulveda Blvd.
Torrance, Calif. 90505
(213) 534-1815
Information on local meetings

■ National Association for Children of Alcoholics
Suite 201
31706 Coast Highway
South Laguna, Calif. 92677
(714) 499-3889
Educational information and referrals for therapy and treatment programs for children of alcoholics

■ *Children of Alcoholism: A Survivor's Manual*, by Judith Seixas and Geraldine Youcha (Crown Publishers, New York, 1985)

■ *Intervention: How to Help Someone Who Doesn't Want Help*, by Vernon E. Johnson (Johnson Institute, Minneapolis, 1986)

FOR EMPLOYERS:

■ Association of Labor-Management Administrators and Consultants on Alcoholism, Inc.
Suite 907
1800 North Kent Street
Arlington, Va. 22209
(703) 522-6272
Information on setting up employe-assistance programs

■ National Institute on Drug Abuse Helpline
(800) 843-4971
Information resource for alcohol and drug problems

Nan Robertson, author of the forthcoming *A.A.: Inside Alcoholics Anonymous*, is a journalist and reformed alcoholic. A *New York Times* reporter for more than 30 years, she won the Pulitzer Prize in 1983 for a personal account of her nearly fatal struggle with toxic-shock syndrome

'I'M ONE DRINK AWAY FROM A DRUNK'

Where would I be without Alcoholics Anonymous? I would be dead. I entered AA in 1975. I haven't had a drink in 12 years. But it took me years to get well, and I had a turbulent recovery, including hospitalization for a nervous breakdown. The most courageous act of my life was not to recover from toxic shock; it was to admit that I was a drunk and to do something about it.

The world is full of functioning alcoholics, and I was one of them. I didn't drink on the job for the longest time. I never lost my job through drinking. I didn't ruin my career. I would have done all of these things had I gone on long enough.

For me, it was a conversation with one person who told me I had to stop, who was not himself an alcoholic but whose father had died an active alcoholic. It was the combination of this man's gentleness and his grief over his father and the fact that he was a doctor—but that really wasn't the important thing. It was that he had suffered through alcoholism. And somehow, what he had to say to me started me on the road back.

I began drinking alcoholically when I was 33, which was about the time I married my second husband, Stanley Levey. I was controlled for the 10 years of our marriage before his death by the fact that he was a moderate drinker, the fact that he loved me. I was careful about my drinking. After he died, very traumatically, all controls were off, and I began drinking suicidally and did so from 1971, when Stan died, almost through 1975, when I went to Smithers (an intensive alcoholism-treatment center in New York City) and then immediately thereafter joined AA.

The worst-kept secret

I drank very heavily at night. My friends at work didn't know this. My editors didn't know this. I thought I'd kept the secret. But when I called up from Smithers joyfully telling everybody I'm an alcoholic, it was the worst-kept secret in the world. My mother had known, all my close friends had known and my stepson had known.

You know, alcoholics drink for every reason they can possibly find. Your life is structured around alcohol. I used to look forward to lunch all the time because there I could have a couple of martinis and a beer. That's pretty pathetic, isn't it? I always wanted to go to restaurants where drinks were served. I didn't want to go to coffee shops. It's very, very typical.

There's a dramatic difference between the way women alcoholics drink and the way men alcoholics drink, which has a lot to do with the way society views women who drink heavily. Women alcoholics generally are hidden alcoholics. It is much less socially acceptable for women to go to bars, to be drunk and disorderly publicly, so very often women drink at home. They often drink alone. Betty Ford is a classic case.

A debt of gratitude

People don't judge people in AA. People don't check up on you. They do not care what you did out there when you were drunk. All they care about is helping you now that you're trying to get sober. That's something that's very hard for outsiders to understand, how extremely flexible AA is. If I feel in crisis now, I'll go to meetings. I still go fairly regularly, but not as much as I did when I was in a terrible state. At most, I would go to AA meetings perhaps five times a week. Now, I go to about one a week, sometimes one every two weeks.

It's very hard, if your life has been saved by an organization—with your help, of course—not to feel deeply grateful to it.

If you are a member of a family that includes an alcoholic, don't protect him or her. Don't call the boss to say, "Fred is sick today and can't come to the office." Don't pour the liquor down the sink. Don't rant and rave. Don't threaten, unless you mean it, that "I'm leaving you."

You have to detach yourself from the person who is an alcoholic and find your own life, because the alcoholic wraps his arms around the bottle—and the family wraps its arms around the alcoholic, and all of their lives become distorted. Their lives are dedicated to the principle that they have to keep this secret and they have to protect their drunk and nobody must know.

What you can do is save your own life. One of the best ways to find help is in Al-Anon, for the families of alcoholics, to know that you're not alone. They teach you to have a life of your own.

The best question to ask yourself or ask about someone you love: Is drinking distorting any part of my life—my working life, my social life, my family life? If it is, then you've got problems. Also, if you are secretly worried about your drinking, chances are you have reason to be worried. All alcoholics are, in their own souls, worried about their drinking. They may deny it; it is a disease of denial. Some people would rather be crazy than be called a drunk. It's still something that people are ashamed of. There's a terrible stigma involved in saying: "I am a drunk. That's what I am. I'm a successful, charming, effective drunk that's sober, and I'm one drink away from a drunk."

Conversation with Beth Brophy

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Economic Costs to Society of Alcohol and Drug Abuse and Mental Illness: 1980

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I. INTRODUCTION AND SUMMARY

A. Background

There are many serious consequences of alcohol and drug abuse, and mental illness. Society suffers the burden of health problems, death, debilitation and incapacitation, crime, motor vehicle crashes, family disruption, pain and suffering and other social problems. Certain aspects of these health problems can be assigned economic values that estimate their impact on our society's economic well-being. The primary emphasis of this report is on identifying and estimating the economic costs to society of alcohol and drug abuse and mental illness (ADM).

There have been several economic analyses of ADM problems. The most comprehensive was a 1981 study by Cruze, Harwood, Kristiansen, Collins, and Jones* in which costs were estimated for 1977. At the time of the research, 1977 was the most recent year for which necessary data were available.

Prior to the 1981 RTI study, a number of monographs had separately analyzed the costs of alcohol abuse or drug abuse or mental illness. These works developed progressively more inclusive estimates of the economic impacts of the disorders, identifying tangible ADM consequences, and using established economic principles to assign monetary values. Each new study improved on its predecessor.

The 1981 RTI study produced the first cost estimates for the three disorders that were generally comparable to each other. Comparability was accomplished in three steps: first, a meticulous review and analysis of the most recent studies on the separate disorders was performed; second, a single consistent methodology for cost estimation was developed; and finally, the estimates of economic cost to society were made.

B. Objectives and Approach

The purpose of this project has been very specific:

> To improve the methodology to update the 1977 estimates of the economic costs of alcohol and drug abuse and mental illness (ADM), to seek better and more comprehensive data sources, and to develop a procedural guide for updating the cost estimates.

---

*Henceforth, the Cruze, Harwood, Kristiansen, Collins, and Jones study will be referred to as the 1981 RTI study.

The 1977 estimates became outdated. They no longer reflected the economic impact of ADM due to the passage of time. One of the first tasks was to select a more recent year for which improved data was available to make cost estimates.

An important part of selecting a more recent year was to thoroughly assess data availability, both for this study and for future cost updates by the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA) or its Institutes. Some costs were not assessed in the 1981 study because appropriate data and analyses did not exist. The availability of data vis-a-vis costs not previously estimated was reassessed. RTI assembled appropriate data on the incidence and prevalence of the ADM disorders and related problems, health services utilization, and productivity and earnings estimates and projections. For each analytic cost category, the project team assessed data availability and currency, suitability of data sets, and frequency of data collection and publication. Based on this assessment, it was recommended that the new estimates be made for 1980. In addition, simplified procedures for updating these cost estimates to 1981, 1982, and 1983 have been developed.

The new estimates are based on and consistant with the concepts and methodology formulated by a Public Health Service task force on cost-of-illness studies (Hodgson and Meiners, 1979). Cost of illness estimates for other illnesses made by the National Center for Health Statistics also employ this methodology, so the cost estimates for ADM can be directly compared with current values for other disorders.

Fundamental improvements have been made in the course of this study. The economic costs of the fetal alcohol syndrome, crime and mental illness, and violent crime due to drug abuse have been added and estimates of productivity of the workforce due to alcohol and drug abuse have been greatly improved.

An advisory panel met in December, 1983 to review the cost estimates from the 1981 study and to identify new data sets and research which could contribute to this project. The discussions also identified a number of aspects of ADM impacts that could not be addressed in this study but which should be addressed in future research. Advisory panel comments have contributed to the improvement of the cost estimates and to the outlining of future directions for ADM cost of illness studies.

C. Results

The economic burden of alcohol abuse, drug abuse, and mental illness in 1980 was an estimated $190.7 billion. Alcohol abuse contributed the largest share of these costs, at $89.5 billion. The expense of mental illness was $54.2 billion, and drug abuse was $46.9 billion. Estimates are broken down by type of cost in table I-1. These values have changed from the 1977 values due to changes in inflation, significant methodological improvements, and population growth factors. The reader is further cautioned that the estimate for mental illness is not completely comparable with those for alcohol and drug abuse because a potentially major impact of mental illness, reduced productivity of the workforce, cannot be estimated at the present time. Comparable values have been estimated for reduced productivity attributed to both alcohol abuse and drug abuse, and these are the largest single components for these two disorders.

Employees with ADM problems are likely to be less productive than otherwise comparable persons. The reduced productivity impact due to alcohol abuse and drug abuse was estimated in this study to be $50.6 billion and $25.7 billion, respectively, or 56 and 55 percent of the total alcohol abuse and drug abuse costs. The estimate of $3.1 billion for reduced productivity due to mental illness appears in table I-1, but it only represents persons reporting partial work disability due to severe emotional or chronic nervous disorders. It does not reflect the costs of the true prevalence of mental illness in our nation.

In contrast, mental illness exacts $18.5 billion due to lost employment (complete disability) of its victims, involving incapacitation either at home or in hospitals. Alcohol and drug abuse have lower costs for lost employment at $4.1 billion and $312 million, respectively.

Treatment services for ADM problems is another major cost category, with a combined value of $31.6 billion, divided among mental illness ($21.0 billion), alcohol abuse ($9.5 billion), and drug abuse ($1.2 billion). This represents direct health services provided to victims of ADM, including long- and short-term hospitalization, services from physicians and other sources.

Table I-1

Costs to Society of Alcohol Abuse, Drug Abuse
and Mental Illness, 1980
($ in millions)

	Alcohol Abuse	Drug Abuse	Mental Illness	Total
Core Costs	$79,607	$29,451	$52,418	$161,476
Direct				
Treatment	9,487	1,200	20,961	31,647
Support	984	243	2,597	3,823
Indirect				
Mortality[a]	14,456	1,980	7,196	23,632
Morbidity[b]	54,680	26,028	21,664	102,372
Reduced productivity	(50,575)[c]	(25,716)[c]	(3,122)[c]	(79,413)
Lost employment	(4,105)	(312)	(18,542)	(22,959)
Other Related Costs	9,919	17,485	1,818	29,222
Direct				
Motor vehicle crashes (property loss)	2,185	d	-	2,185
Crime[b]	2,347	5,910	870	9,127
Public	(2,062)	(4,454)	(635)	(7,151)
Private	(261)	(1,345)	(235)	(1,841)
Property loss/damage	(24)	(111)	(-)	(135)
Social welfare program	38	2	201	241
Other	2,912	537	659	4,108
Indirect				
Victims of Crime	172	845	-	1,017
Crime careers	-	8,725	-	8,725
Incarceration	1,801	1,466	88	3,356
Motor vehicle crashes (time loss)	464	d	-	464
Total	$89,526[c]	$46,936[c]	$54,236[c]	$190,698

Totals may not add due to rounding.

[a] At 6 percent discount rate. As suggested by the PHS Guidelines document, the present value of lost future productivity due to premature mortality was also calculated using discount rates of 10 and 4 percent. The use of a 10 percent rate decreases indirect costs by the following amounts: alcohol abuse--$4,881 million; drug abuse--$704 million; and mental illness--$2,444 million. The use of a 4 percent rate increases indirect costs by the following amounts: alcohol abuse--$4,455 million; drug abuse--$638 million; and mental illness--$2,177 million.

[b] Components are indicated in parentheses.

[c] The total costs to society for each of the three ADM disorders are not comparable, since the completeness of data available for each cost category varied significantly. For example, the estimate of reduced productivity is relatively complete for alcohol abuse, only partially complete for drug abuse, and incomplete for mental illness.

[d] Although costs are hypothesized to occur in this category, sufficient data are not available to develop a reliable estimate.

Source: Research Triangle Institute.

Premature mortality is another serious consequence of these disorders, resulting from drug overdoses, liver disease, suicide, homicide, motor vehicle crashes and other causes. The value for alcohol abuse was $14.5 billion, of which $5.9 billion was motor vehicle deaths caused by alcohol abuse. Cirrhosis of the liver represented $3.4 billion and homicide $2.4 billion. Drug abuse cost $2.0 billion (for accidental overdoses), and mental illness cost $7.2 billion (mainly for suicide). These values were estimated at the six percent discount rate.

Motor vehicle crashes due to alcohol abuse have a number of different cost impacts. In addition to mortality (mentioned above), there are property losses ($2.2 billion), time losses of motorists ($464 million), and assorted public criminal justice and highway safety expenditures.

Alcohol abuse is also strongly related to violent crime. Criminal justice system costs ($2.3 billion), incarceration losses ($1.8 billion), and victim of crime losses ($172 million) were due to alcohol involvement in the personal victimizations of assault and homicide, and in property crimes such as robbery, burglary, and larceny.

The involvement of drug abuse in crime carries extensive economic costs. Crime careers (drug trafficking, property crime, and various consensual offenses) motivated by drug addiction were estimated to cost society $8.7 billion because addicts pursued socially non-productive careers. Additional costs were public and private criminal justice expenses ($5.9 billion), lost employment of crime victims ($845 million) and the ultimate incarceration of convicted criminals ($1.5 billion). These costs do not include the value of illicit drugs consumed, estimated by various sources at between $9 and $74 billion annually.

Mental illness is also related to crime as is apparent from the costs of public and private expenditures ($870 million) and incarceration. These costs are dissimilar to the values for alcohol and drug abuse, however. It is contended that deinstitutionalization of many mentally impaired persons has made them a burden on the criminal justice system due to public order offenses of vagrancy and disorderly conduct, and these costs have been estimated for this study. There is still a lack of consensus on the role of mental illness and violent crime, and these costs have not been estimated.

D. Comparison With Previous Study

1. General

Economic cost studies of ADM should not, in general, be used as indicators of changes in the severity of these disorders over time. While these estimates do convey important knowledge of the relative magnitude of the problems at a point in time, there are many changes over time that make interpretation of decreases and increases difficult. One of these factors is inflation in prices and wages, which may change at different rates for different components. Other factors that may contribute to interpretation of cost changes over time include changes in the true prevalence of the problem, population growth, and the age and sex distribution of the population who suffer from these problems.

Changes in the severity and nature of alcohol and drug abuse are routinely monitored in our society. The National Institute on Alcohol Abuse and Alcoholism (NIAAA) and the National Institute on Drug Abuse (NIDA) sponsor recurrent surveys on the incidence, prevalence, and impacts of alcohol abuse and drug abuse in our society. Both series use survey methodologies that are consistent from year to year, making these appropriate vehicles for monitoring trends in the levels and severity of the two disorders.

The trend in problems from alcohol abuse was constant during the 1970s. The most recent study in the series concluded that there was no indication that the level or severity of alcohol problems in the United States changed throughout the 1970s. The prevalence rates of problem drinking and its specific consequences remained relatively constant over the period studies. However, the total magnitude of the problem has increased with population growth.

Throughout the 1970s there was an upward trend in the use of psychoactive substances. Analysis has indicated that drug use peaked in 1979 and that there was a statistically significant decline in use between 1979 and 1982.

The level and severity of mental illness in our society have not been followed on as regular a basis. However, the National Institute of Mental Health has recently begun the Epidemiologic Catchment Area surveys (Eaton, Regier, Locke, and Taube, 1981), which may ultimately yield methodologies and data useful for making periodic estimates of the prevalence of mental illness as well as alcohol abuse and drug abuse in the United States.

Table I-2

Change in Cost to Society of Alcohol Abuse, 1977-1980
($ in millions)

Cost Items	Value	Share of Change (percent)
Total in 1980	$89,526	
Total in 1977	49,374	
Change 1977-1980	40,152	100.0
Change due to		
Inflation	15,286	38.1
New methodology for		
Reduced productivity	18,872	47.0
Fetal alcohol syndrome	3,236	8.1
(Subtotal, changes above)	(37,394)	(93.1)
Other changes	2,758	6.9

Source: Research Triangle Institute

2. Changes in Specific Cost Components

The economic burden on society of ADM was an estimated $150.7 billion in 1980. To identify differences between the 1977 and 1980 estimates, each 1977 cost component estimate was adjusted upward by an appropriate price index to reflect 1980 dollars. Using this methodology, inflation between 1977 and 1980 was estimated at $32.872 billion for the three disorders. This increase (38.8 percent) was the largest single factor in the three-year change.

No cost change is authoritatively attributed to change in the incidence, prevalence, or severity of the disorders. However, improvements in knowledge about the disorders have made it possible to include the value of impacts that were omitted from the 1977 estimates. Among these, the largest were reduced productivity in the work force due to alcohol abuse and drug abuse. These contributed 22.3 percent and 26.0 percent of the $84.7 billion increase, respectively. Other new estimates, changes in methodology, and new data sources accounted for the balance of the change between 1977 and 1980; within this group, the addition of estimates for fetal alcohol syndrome was the largest factor, accounting for 3.8 percent of the total 1977-1980 increase.

The total cost of alcohol abuse and alcoholism in 1980 was estimated at $89.5 billion. The 1977 estimate was $49.4 billion. The difference of $40.1 billion was almost completely due to inflation and a change in the methodology for estimating the effect of alcohol abuse in the workforce (table I-2). Inflation accounted for $15.3 billion of the total increase, or 38.1 percent.

Productivity losses in the economy were the largest source of change in total costs with growth over inflation of the 1977 value at $18.9 billion (47.0 percent of total growth). New analyses performed as part of this study found that alcohol abuse affects productivity by more than had been previously estimated. It is now estimated that problem drinking reduces individual productivity 21 percent below that of otherwise similar persons. The prior estimated impact was a 14 percent reduction.

Inclusion of fetal alcohol syndrome (FAS) in the study was the other major change in methodology, accounting for cost increases of $3.2 billion (8.1 percent of the total increase). FAS is a serious combination of birth defects that researchers have linked to maternal alcohol use during pregnancy.

Both physical and mental defects are involved. In addition to the health treatment required by FAS victims, the associated mental impairment requires special education, training, and support services throughout their lives. The mental impairment causes reduced productivity and in some cases institutionalization.

These three factors--inflation, reduced productivity in the workforce, and fetal alcohol syndrome--account for 93 percent of the differences between the 1977 and 1980 estimates. Of the remainder, 3 percent can be attributed to population growth. The final 4 percent is due to other minor changes.

Cost estimates for drug abuse in 1980 are $46.9 billion, a 286 percent increase over the 1977 estimate of $16.4 billion. Simply inflating the 1977 estimates would have led to an increase of $5 billion (16.4 percent of the total increase). All changes are itemized in table I-3.

The change of $22 billion resulted from findings in this study that high levels of marijuana abuse are related to reduced productivity in the workforce. Although the problem had been suspected, it had not been rigorously studied previously. It was found that persons who had ever used marijuana daily for a month or longer had household incomes 27.9 percent lower than persons with otherwise similar characteristics.

Violent crime has been increasingly linked to illicit drugs. Costs due to this problem have been included in this study for the first time. The violent crime-drug link is estimated to have cost society $1.3 billion in 1980 (4.4 percent of the increase).

The remaining change of $2.2 billion (7.2 percent) is due to changes in a number of different components, including the effect of 3 percent population growth.

The costs of mental illness grew from $40.3 billion to $54.2 billion, 34.6 percent over the three-year period 1977-1980. Virtually all of the growth was due to inflation. Merely adjusting the 1977 figures for inflation accounts for 90.2 percent of the increase (table I-4). There were changes in two components that bear further discussion. The value for reduced productivity increased by $962 million over inflation. The assumed level of impairment of the partially disabled mentally ill was increased from 14 percent to 24 percent.

Table I-3

Change in Cost to Society of Drug Abuse, 1977-1980
($ in millions)

Cost Items	Value	Share of Change (percent)
Total in 1980	$46,936	
Total in 1977	16,387	
Change, 1977-1980	30,549	100.0
Change due to		
Inflation	5,010	16.4
New methodology for		
Reduced productivity	22,003	72.0
Violent crime	1,338	4.4
(Subtotal, changes above)	(28,351)	(92.8)
Other changes	2,198	7.2

Source: Research Triangle Institute

Table I-4

Change in Cost to Society of Mental Illness, 1977-1980
($ in millions)

Cost Items	Value	Share of Change (percent)
Total in 1980	$54,236	
Total in 1977	40,287	
Change, 1977-1980	13,949	100.0
Change due to		
Inflation	12,576	90.2
New methodology for		
Reduced productivity	962	6.9
Social welfare administration	-506	-3.6
(Subtotal, changes above)	(13,032)	(93.4)
Other changes	917	6.6

Source: Research Triangle Institute

The cost of administering social welfare benefits to the mentally ill declined by $506 million, or about 3.6 percent of the total increase. This major change occurred because better data about the cost of these programs were used and not because benefits or levels of service changed.

The major increases in costs between the 1977 and 1980 studies are due primarily to changes in methodology, or to inflation in wages and prices and population growth. The authors do not attribute any of the growth in costs to changes in the incidence, prevalence, or severity of ADM. Making such a determination is extremely difficult due to the large number of different cost impacts studied and the many different data sets used. Because many of the values used in preparing cost estimates are derived from sample estimates, sampling error must be taken into account if costs are to be compared. That task is beyond the scope of the present effort.

E. Organization of This Report

The following chapter (II) provides an introduction to the concepts involved in estimating the economic costs of ADM and of all illnesses in general. Specific topics discussed include prior ADM cost studies, the nature of economic costs (versus transfers), direct versus indirect costs and double counting. An examination of recent findings about the prevalence of alcohol and drug abuse and mental illness follows this discussion.

The majority of this report (chapter III) describes the methodology used to estimate costs in this project. Because this project is primarily an update of a prior study, the text focuses on any improvements on or modifications of the previous study's methodology and does not offer detail about the rationale for including particular costs or the specific computations performed. Details not found here are documented in the 1981 study.

There are strong implications of cost of illness studies for evaluations of public interventions. These implications are explored in chapter IV in a discussion of the application of cost benefit and cost effectiveness analysis to ADM public policies. Suggested areas for further research in ADM cost-of-illness studies are discussed in the last chapter. A series of appendices that present selected details of the application of the cost-of-illness approach to ADM problems concludes this report.

A protocol for producing updated cost estimates for ADM also appears in the appendix. The protocol has been used to calculate values for 1981, 1982, and 1983, which are included in the appendix.

Perhaps the most significant contribution of this report is the rigorous analysis of the impact of alcohol and drug abuse on productivity in the economy. The largest cost elements in this study, the productivity impacts of ADM, are very significant. A full discussion is included in the appendix.

Another major impact of ADM, the fetal alcohol syndrome, is analyzed in this report for its economic implications. Evidence on its incidence and health impacts is presented in the appendix. At the outset of this study, it was hoped that cost estimates could be made for the impact of alcohol abuse on neoplasms of the gastrointestinal tract. Knowledge about these health problems has grown, but it is premature to make cost estimates. The implications of attributing all or some of these neoplasms to alcohol abuse are explored in a section of the appendix.

The relationship of crime to ADM was more carefully analyzed in this report than in the earlier study. Recent findings indicated that the methodology with respect to mental illness and criminal justice system costs, as well as drug abuse and violent crime, required change. These changes are discussed. There is additional discussion of the relationship of drug abuse to property crime. In addition, a number of selected topics relating to the impacts of ADM on the economy, including social welfare programs (the value of program transfers, and the costs of administering programs) and expenditures on illegal drugs and alcohol, are discussed in the appendix sections.

II. CONCEPTUAL PRINCIPLES FOR COST OF ILLNESS STUDIES

Alcohol and drug abuse and mental illness are disorders which pervade every level of our society, with many different manifestations in all age groups - infants, children, adults, and the elderly. It has been estimated that up to 20 percent (Regier, Goldberg, and Taube, 1978) of the adult population experience these problems during a year. The dimensions of the disorders seem limitless - causing thousands of deaths and legal, personal, and occupational problems and requiring enormous amounts of health-care resources.

One particular dimension of these disorders, the economic cost, has been singled out for study because it offers a framework for integrating many of the problems. The economic framework offers a manner of valuing the real resources that are affected by the disorders and makes it possible to make comparisons between different health problems at a point in time or a single health problem at different times.

A. Background

One of the earliest cost-of-illness studies was performed by Rashi Fein (1958) on the cost of mental illness. At that time, he estimated that the direct and indirect costs were well in excess of $3 billion. However, this was considered a conservative estimate since many costs could not be calculated due to data limitations.

The pioneering work by Fein was further developed by Rice (1965, 1966) in her studies of cardiovascular illnesses and, subsequently, all illnesses. Rice's cost analyses laid a solid methodological base on which more recent estimates of the costs of illnesses (notably, Cooper and Rice, 1976, and Paringer and Berk, 1977) have been built.

Rice (1966), Cooper and Rice (1976), and Paringer and Berk (1977) estimated the costs of mental illness. These studies indicated that the costs of mental illness were close to 10 percent of the nation's health expenditures. Each of these studies included some of the costs of alcohol and drug abuse in addition to the other clinically diagnosed mental disorders, but these costs were not distinguished. The unioue nature and problems associated with alcohol and drug abuse motivated the performance of separate cost-of-illness studies for the two disorders.

A Research Triangle Institute (Rufener, Rachal, and Cruze, 1977) study of drug abuse indicated that high proportions of actual economic costs were not reflected in the traditional cost-of-illness framework. The authors identified cost components related to manifestations of trauma (e.g., drug overdoses), employment problems, and large nonhealth social costs (e.g., disability and support payments and criminal justice costs), and crime careers.

The economic costs of drug abuse had been previously assessed in examinations by Lemkau, Amsel, Sanders, Amsel, and Seif (1974), Rufener et al. (1977), and Goldman (1978). A number of manifestations of drug abuse were included in these studies, such as the more general categories of mental illness, different kinds of trauma (accidental overdoses and suicidal overdoses of drugs), and activities outside the health sector. As with alcohol abuse, many of the impacts of drug abuse involve crime.

A study of the costs of alcohol abuse by Berry and Boland (1977) identified large cost components due to alcohol abuse that had been included previously under illnesses other than mental disorders, or that had been entirely ignored by the health cost studies. Examples of these misclassified or ignored costs included alcohol abuse-related trauma (accidents, violence and poisoning), productivity losses in the work place, and other nonhealth social costs.

Levine and Levine (1975) gave more detailed analyses of the economic cost of mental illness than that offered by Rice (1966), Cooper and Rice (1976) and Paringer and Berk (1977). Levine and Levine used essentially the same estimation approach as the studies of all illnesses, although they looked beyond the formal mental illness-related diagnostic codes to examine the costs and relationship of suicide (which in cost-of-illness studies is traditionally classified as trauma along with accidents, violence and poisoning). However, there was no identification of mental illness costs associated with alcohol abuse and drug abuse.

The first study to make comparable, consistent economic cost estimates for alcohol and drug abuse and mental illness was performed by the Research Triangle Institute (Cruze et al., 1981). This cost analysis started with a thorough analysis of the three principal cost studies for each disorder (mentioned immediately above), examining them for methodology, cost framework, conceptual development and data sources. The three studies were carefully compared for substantive differences in approach, and the reasons for these differences were noted. In particular, they were compared with respect to a document produced by the Public Health Service on guidelines for cost-of-illness studies (Hodgson and Meiners, 1979). The guidelines document established an analytical framework, laid out conceptual and methodological issues, and recommended procedures for cost estimation that were all intended to assist researchers in producing cost studies that were consistent and readily comparable.

The study by the Research Triangle Institute developed a methodology for estimating costs of the three disorders that was consistent with the guidelines and produced comparative cost estimates for the three problems. This methodology was reviewed and accepted for implementation by an advisory panel from the institutes of ADAMHA.

Other key features of this study were identification and documentation of alternative cost estimation methodologies, analysis of further interactions of alcohol and drug abuse and mental illness with the economy, and identification of cost components of the three disorders for which there were insufficient data to make estimates. Cost overlaps between the three disorders were identified and eliminated from the final cost estimations. In addition, cost components were specifically defined so that the estimates could be compared with each other, and with estimates for other illnesses.

B. Conceptual Framework

A number of conceptual issues must be addressed regardless of the specific procedures used to develop estimates of the economic costs to society of alcohol and drug abuse and mental illness (ADM). These are discussed in two parts. The first outlines an overall conceptual framework for developing cost-of-illness estimates, moving from general principles of cost and cost-of-illness measurement to the application of these principles to ADM problems. The second section discusses five specific conceptual issues that must be addressed in estimating the economic costs to society of ADM problems.

1. Principles for Economic Analysis

The first step in developing an overall framework for measuring the costs of ADM disorders is to select the basic theories and concepts for measuring these costs. A basic assumption of the economic theory underlying the concepts used in this study is that resources are finite or "scarce."

Therefore, when resources are allocated to the production of certain goods and services, they are consumed and cannot be used in the production of other goods and services. The loss of the opportunity to use resources for another purpose is the "opportunity cost."

Economic transfers, as the name implies, do not represent a consumption of resources per se but only the shift in control over the use of resources from one segment of society to another. Because they are distinct from economic costs, it is inappropriate to include transfers in an estimate of the economic cost to society of an illness. Previous estimates of the value of property stolen due to drug abuse, for example, have been as high as $6.2 billion per year. Although these losses are legitimate costs to the individuals from whom the property is stolen, from society's perspective the property is transferred from one individual to another and no social cost is incurred. However, resources are used to administer property insurance programs, and resources are required to replace lost or damaged property. Procedures for estimating these real costs will be detailed in this study.

Economic or opportunity costs may be measured from various perspectives. Employers incur costs when treatment is provided for employees with ADM or other health problems at company expense or when production is lost due to factors such as lowered productivity or absenteeism. Costs are borne by an entire society as current resources are used for prevention, treatment, and rehabilitation and as potential resources are lost due to lower productivity, absenteeism, or withdrawal from the workforce because of death or disability. Since the primary purpose of this study is to provide information for policy making and resource allocation decisions within ADAMHA and other government agencies, costs from the perspective of our entire society are the most relevant and will be explored in this study.

Costs are either direct or indirect. A direct or explicit cost is one in which resources are consumed, and a formal payment is made in cash or in kind (i.e., through the direct provision of some commodity or service). When resources are used to treat an ill person, the labor is paid for in wages, the materials used are paid for as direct purchases, and the capital used is paid for in principal and interest payments. On the other hand, an indirect or implicit cost is one in which no formal payment is made for resources used. For example, hospital patients are not paid for the value of the goods and services they would have produced had they not been in treatment. Indirect costs include the value of foregone production of household goods and services. When alternative resources (e.g., housekeepers, babysitters, launderers) are hired to supply household services to family members, the foregone production is a cost.

Indirect costs are frequently incurred over a period of time. The principal example of this is foregone production due to premature death. In this situation, the lost stream of future earnings is counted as a cost in the year the death occurs. This technique reduces or "discounts" the value of a dollar in a future time period into an appropriate value for the current time period.

Illness and disease frequently cause a wide variety of psychosocial problems whose cost cannot be readily measured in monetary terms. Social costs include loss of a body part or speech, disfigurement, anticipation of death, pain, and grief. In addition, the environment created by an illness reduces self-esteem and feeling of well-being and induces anxiety, resentment, and emotional problems that often require psychotherapy. Problems of living may develop, leading to family conflict, antisocial behavior, or suicide. Despite rehabilitation efforts, the quality of life enjoyed before the illness may not be restored. Although social factors are important components of the total burden of an illness, appropriate measures of these social costs are not well developed. The focus of this study, therefore, is on estimating the economic costs which can be readily quantified in dollars. These will be referred to as economic costs to society throughout the remainder of this report.

2. Principles for Cost-of-Illness Studies

In applying the above general principles to the estimation of the cost of an illness, it becomes apparent that the costs of an illness or disease may be divided into two major categories: "core costs," primarily those occurring in the health sector, and "other related" costs. Each category contains both direct and indirect costs.

As outlined in the PHS guidelines document on cost-of-illness studies (Hodgson and Meiners, 1979), direct core costs are the costs of diagnosis, treatment, continuing care, rehabilitation, and terminal care for illness and trauma. These include expenditures for hospitalization; outpatient clinical care; nursing home care; home health care; services of primary physicians and specialists, dentists, and other professionals; drugs and drug sundries; rehabilitation counseling; and other rehabilitation costs to overcome illness-related impairments.

Indirect core costs result from losses in output as time is lost from paid work, or household services are not provided because of treatment, injuries, premature mortality, or other reasons. As indicated above, the measures of output loss are foregone earnings and the imputed market value of unperformed housekeeping services. In addition to extended time lost from work, indirect core costs may also be incurred when an illness lessens productivity on the job or causes excessive absenteeism.

Other related direct costs in the nonhealth sector which must be borne by patients and other individuals include costs of: transportation to health providers; hiring household help for cleaning, laundering, cooking, and babysitting; special diets; items for rehabilitation and comfort; and vocational, social, and family counseling services. These direct costs may also include the value of property damaged or destroyed in vehicular accidents or crimes caused by drug or alcohol abuse.

Examples of other related indirect costs include the opportunity cost of time spent in prison or pursuing a criminal career and of time spent by family members and friends in visiting hospital patients, their physicians, or other health professionals.

3. Application of Concepts to ADM Problems

The concepts summarized above may be readily incorporated into a conceptual framework to estimate ADM costs. Direct core costs include primarily the costs of treatment for the various ADM problems. In the case of mental illness, these costs are incurred as a direct consequence of the illness as individuals seek treatment in public and private mental health treatment facilities. In the case of alcohol abuse and drug abuse, these costs are incurred as individuals seek treatment for problems directly and indirectly caused by their substance abuse. In addition, the direct core costs include costs for such supportive activities as prevention and the various research, training, and administrative functions that are carried out by both public and private agencies to support ADM treatment activities.

Indirect core costs for ADM problems arise primarily from morbidity or premature mortality, measured in terms of both foregone earnings and the imputed market value of lost household services. Additional indirect costs may occur from lost work or household production due to outpatient or inpatient treatment or from lowered productivity for those with ADM problems who continue to work or perform household activities.

Other related direct costs for ADM problems are quite diverse. The two largest categories of these costs stem from ADM-induced crimes and from property destruction (e.g., due to vehicular accidents or crime). In addition, other related direct costs include an appropriate share of the administrative costs of various social welfare programs providing services to individuals with ADM problems.

Other related indirect costs involve time and productivity losses not directly related to the health of the ADM victim. There is a very significant cost for drug abusers whose crime careers and incarceration take them away from productive activities and constitute a loss for society as a whole that is causally related to drug addiction or to the drug trade. This cost component is not very large for alcohol abuse and mental illness.

A summary of the specific cost elements suggested for use in cost-of-illness studies is provided in table II-1. These costs are organized according to the core costs/other related costs framework presented in the PHS guidelines document.

C. Specific Issues

1. Definitions

By definition, ADM exists when a diagnosis of the disorder is made by a health professional using professionally accepted criteria. Established diagnostic systems include the International Classification of Diseases - 9th Edition (ICD-9) and the Diagnostic and Statistical Manual III (DSM-III). Most health system diagnostic data are now maintained using the ICD-9 system. There is a cross-reference to data that were coded using earlier editions. The use of DSM-III is advocated by mental health professionals because there are specific codes and criteria for alcohol and drug abuse and mental illness. The ADM diagnostic conditions used in this study are based on ICD-9 and are specified in tables III-4, III-7, III-8, and III-9. The ADM-specific conditions are primarily those in the large category of mental illness (codes 290-316). Because ADM effects many aspects of health, other conditions are defined as "ADM-related." These are completely or partially attributed to ADM in the cost estimates. Notable examples are liver cirrhosis, drug overdoses, suicide, and other trauma.

Table II-1

Cost Elements for Cost-of-Illness Studies

I. CORE COSTS

   A. Direct
      1. Hospitalization
      2. Outpatient clinical care
      3. Nursing home care
      4. Home health care
      5. Services of primary physicians, specialists, and other professionals
      6. Drugs and drug sundries
      7. Rehabilitation counseling
      8. Rehabilitation aids

   B. Indirect
      Lost productivity due to:
      1. Mortality
      2. Morbidity (lost productivity)
         a. Reduced productivity
         b. Lost productive time

II. OTHER RELATED COSTS

   A. Direct

      1. Transportation
      2. Household expenditures and help for the household
      3. Special equipment or alteration of property for rehabilitation or comfort
      4. Counseling, retraining, and reeducation
      5. Property losses (destruction, for instance, from vehicular accidents or criminal activity)
      6. Criminal justice system
      7. Welfare system administration (not the transfers themselves)

   B. Indirect

      1. Lost productivity of family and friends
      2. Lost productivity resulting from incarceration due to ADM problem
      3. Lost productivity from full-time pursuit of a crime career in order to support a drug habit
      4. Lost productivity of persons other than crash victims due to ADM-caused motor vehicle accidents

Source: Derived from Hodgson and Meiners (1979).

Consequences of ADM outside of the health sector have been determined based on established causal links between ADM (chronic or episodic) and tangible consequences (for example, drinking and driving, and drugs and crime).

2. Incidence and Prevalence

In estimating these costs to society, the distinction between incidence and prevalence of ADM has been carefully made. Strictly speaking, incidence of an ADM problem is defined as the number of new cases of a problem that occur in a given period of time. Prevalence refers to the total number of cases at a point in time or over a given period of time. Since the purpose of this study has been to estimate the costs to society of ADM problems, we were primarily interested in the prevalence of each of the ADM problems for a specific time period (i.e., 1981).

It would be interesting to examine the pattern of costs caused by an individual's problem over time from a benefit/cost perspective. The benefits derived from successful treatment of a problem could then be subtracted from the costs. However, incidence data are not readily available, and the primary emphasis of this study is on prevalence.

3. Association and Causality

Behaviors may be caused by ADM problems or they may merely be associated with them. The importance of distinguishing between cause and association has been pointed out in previous efforts. In addition, the literature review conducted for the previous RTI study indicated the difficulty in establishing valid measures of causality. Despite these problems, this study has attempted to estimate costs to society by using measures of causality rather than correlation or association. Wherever possible, the results of previous studies that have used appropriate designs and analytic techniques to develop estimates of the degree of causality were incorporated into the estimation procedure. Nonetheless, the ideal of establishing causality could not always be achieved due to current state-of-the-art limitations. When no reasonable causality estimate was available, rational assumptions and indirect statistical techniques were used to approximate the extent of causality. Whenever simple associations were relied upon, that fact is noted.

4. Foregone Earnings

As indicated earlier, a critical input to estimating the indirect costs of ADM is the value of foregone earnings of individuals with ADM problems who die prematurely or who for other reasons become unproductive. In order to develop a consistent approach to estimating the value of this cost component across the three ADM problem areas, a single set of earnings profiles for the general population subgroups (e.g., classified by age, sex, race, educational attainment) was used. The profiles were then adjusted to reflect the demographic characteristics of the populations with each of the three ADM problems. Choices have been made on the use of adjustments for expected labor force participation and unemployment for populations with ADM problems and for a less than full employment national economy, discount rates for future earnings values, and rates of labor productivity growth. Additional decisions were made on the conceptual bases for estimating the costs of lowered on-the-job productivity due to ADM problems, the costs of foregone household production by individuals not in the officially-defined labor force, and the value of production lost when individuals volunteer to work in the ADM treatment/prevention system.

Recently developed unpublished data on the present values of age/sex-earnings profiles prepared by the staff of the National Center for Health Statistics provided an appropriate source for estimating many of these indirect costs. The assumptions underlying the development of these profiles were carefully reviewed, and the resulting profiles were found to be appropriate for use in this study.

5. Double Counting

The final problem addressed was that of double counting the costs of a given condition in developing the total cost of ADM problems. This might happen, for example, when foregone earnings are counted in the general estimate of premature death from alcohol abuse and again in specific estimates of traffic fatalities. This double counting might also occur in situations in which an individual with an ADM problem (e.g., an alcoholic) is treated in a specialty setting that is not organized primarily to treat the problem (e.g., a Community Mental Health Center). This person's costs would be counted twice in estimating the costs to society of ADM problems (e.g., once as an alcohol abuse cost and again as a mental illness cost). Such situations should be identified and the double counting eliminated in developing a composite estimate of the costs to society of ADM. One possible approach is to specifically identify the various specialty settings in which more than one ADM problem is treated and the proportions of treatment resources used to treat the separate ADM problems in each of these specialty settings. This procedure enables us to develop estimates of both the treatment costs for each ADM problem and the treatment costs for each type of specialized ADM facility.

Double counting may also occur in situations where it is difficult to specify to which of the three ADM problems a particular cost element should be assigned. Where, for example, should the costs for the treatment of an individual with more than one ADM problem be assigned? The costs might be reported as an unallocated total and not be included in the cost estimates of the separate ADM problems, or they may be assigned to a single problem (such as the primary diagnosis) or to multiple problems according to appropriate procedures. A second example is the difficulty in assigning the costs of administration of an agency concerned with more than one ADM problem. Here, too, the costs might be assigned to an unallocated total and not to separate ADM problems, or they can be assigned to one ADM problem by some rule. A third alternative is to allocate them to the separate ADM components in proportion to other direct agency costs.

D. Definition of ADM

One of the most difficult facets of this economic cost study is defining the nature of alcohol, drug abuse, and mental illness in order to discern the incidence or prevalence rates of these disorders and in order to estimate their economic impact.

Mental disorder is generally defined to include alcohol abuse and alcohol dependence, as well as drug abuse and drug dependence. The large category in the International Classification of Diseases, "Mental Illness," includes specific subcategories for alcoholic psychoses, alcoholism, and alcohol abuse, as well as drug abuse, and drug dependence. A widely used classification system, DSM-III, gives distinct diagnostic criteria for diagnoses of alcohol abuse, alcohol dependency, drug abuse, and drug dependency.

One of the greatest challenges in performing an economic cost study is in clearly differentiating among diagnoses, but it is not uncommon for an individual to suffer from more than one mental disorder at a time. For example, schizophrenics may also abuse alcohol and/or drugs. Where it is desirable to differentiate between alcohol abuse/dependence, drug abuse/dependence, and other mental disorders, the definitional problems become enormous.

The distinctions among multiple disorders are a major problem in the fields under study. Nonetheless, an attempt to differentiate among the different types of disorders was made in this study, and the results reported in the balance of this report reflect our best attempts. Fortunately, the diagnostic data typically used in studies of these populations usually distinguish between the disorders. Unfortunately, although patients or individuals whose ADM problems have economic impacts usually have a primary diagnosis, they may also have additional diagnoses secondary to the primary cause or equivalent to it. In some data sets, multiple diagnoses are made, with no indication of their order of importance.

In this study, an individual's "principal" problem is defined as the "primary" diagnosis or, if there is no priority given, the "first listed" diagnosis. The cost estimates made for alcohol abuse, drug abuse, or other mental disorders using this rule are somewhat arbitrary. For the present, these imprecisions are unavoidable.

E. Prevalence of ADM

Recent studies have estimated an unduplicated count for alcohol abusers, drug abusers, and other mental illnesses at 15 percent of the population. A study by Regier et al. (1978) estimated that 15 percent of the U.S. population, including individuals of all ages, experienced alcohol and/or drug abuse problems or a mental disorder during 1975. While it excluded alcohol and drug abusers who received treatment only in clinics specializing in these disorders, it included people with alcohol and drug abuse problems seen in other specialty mental health sectors and in the general health sector. It was estimated that 6.7 million persons were treated in the specialty mental health sector, 1.1 million were inpatients in general hospitals or nursing homes, and 19.2 million received outpatient services in the general health sector. Another 6.9 million individuals were judged to have not received treatment. These estimates included adjustments to avoid duplications for individuals seen across different settings and sectors.

The epidemiological catchment area (ECA) studies supported by NIMH have yielded data supporting the 15 percent prevalence estimate. The ECA studies use the recently developed Diagnostic Interview Schedule (DIS) which contains information necessary to make DSM-III diagnoses of specific mental disorders. Myers et al. (1983) estimated the 12-month prevalence rates of DIS/DSM-III psychiatric disorders to be 12 to 13 percent in three communities over a 6-month period, and 13 to 15 percent over a 1-year period (see table II-2). These prevalence estimates exclude values for phobia and dysthymia but include values for alcohol abuse/dependence and drug abuse/dependence. The estimates of the prevalence of alcohol abuse/dependence and drug abuse/dependence made in this study are presented in table II-2. By averaging the rates for the three different communities, the prevalence of mental illnesses, including phobias, was estimated at 18 percent over a 6-month period. Excluding phobias, the average was 12.6 percent. Again using the diagnostic criteria as specified in DSM-III, the rate of alcohol dependency in the three communities was 3.2 percent, alcohol abuse was 1.9 percent, drug dependency was 1.0 percent, and drug abuse was 1.1 percent. Neither these nor other estimates based on ECA studies have adjusted for persons with multiple disorders.

Similar work has resulted from epidemiological studies of alcohol abuse and drug abuse. General population surveys have indicated significant prevalence rates for each of the disorders, although they have not yet attempted to identify overlaps between the respective populations.

The prevalence of problem drinking, or alcohol abuse/dependence, has been estimated to be about 10 percent of the adult population. A series of national household surveys conducted since the middle 1960s has generated prevalence estimates utilizing a variety of diagnostic criteria. The most recent survey (Clark and Midanik, 1982) estimated that 10 percent of the adult population exhibited symptoms of loss of control while drinking or dependency on alcohol during the survey year and that 5 percent of the adult population attributed at least one social consequence to abuse of alcoholic beverages.

The prevalence of specific problems associated with drinking in national surveys conducted in 1967 and 1979 is presented in table II-3. The national surveys have demonstrated that a wide variety of problems are associated with alcohol abuse. For both national surveys, the most prevalent problems were psychological dependence and symptomatic drinking. Other problems include health problems, job problems, belligerence, difficulties with the law or one's spouse, and binge drinking.

Table II-2

Six Month Prevalence of Mental Illness, Alcohol Abuse/Dependence and Drug Abuse/Dependence in Three Communities

	New Haven	Baltimore	St. Louis	Simple Average
Any Mental Illness[1]	16.9	22.6	14.8	18.1
Any Mental Illness [2]	13.2	12.9	11.6	12.6
Alcohol Abuse/Dependence	4.8	6.1	4.5	5.1
Alcohol Dependence	2.8	4.2	2.6	3.2
(Alcohol Abuse)[3]	(2.0)	(1.9)	(1.9)	(1.9)
Drug Abuse/Dependence	1.8	2.3	2.2	2.1
Drug Dependence	1.1	0.8	1.1	1.0
(Drug Abuse)[3]	(.7)	(1.5)	(1.1)	(1.1)

[1] Excludes dysthmyia (DSM-III, an episode of depression that does not meet diagnostic criteria for "Major Depression").

[2] Excludes dysthymia and phobia (large variation in prevalence estimates for phobia were obtained from three sites, suggesting caution in use of its estimates).

[3] This value is the difference between the two prior values.

Source: Myers, Weisman, Tischler, Holzer, Leaf, Orvaschel, Anthony, Boyd, Burke, Kramer, and Stoltzman (1983)

Table II-3

Prevalence of Problems Associated with Drinking in the Past 12 Months for Males and Females in the 1967 and 1979 National Surveys (in Percentages)

Problem Area	1967 Survey			1979 Survey		
	Male	Female	Total	Male	Female	Total
Health problems	6	5	5	4	2	3
Belligerence associated with drinking	5	3	4	8	4	6
Problems with friends	2	*	1	3	1	2
Symptomatic drinking	11	5	8	20	9	14
Psychological dependence	49	29	37	26	17	21
Job problems	3	2	2	7	2	4
Problems with the law, police, accidents	1	*	*	2	1	1
Binge drinking	1	*	1	1	*	1
Problems with spouse**	1	0	1	2	*	1
(N)	(751)	(608)	(1,359)	(762)	(1,010)	(1,772)

Note: The percentages are weighted figures and may not total to 100 percent due to rounding; totals shown are the actual number of cases. Slight variations in these totals occur because of nonresponse, etc.

*Less than 0.5 percent.

**Last 2-1/2 years for National 1967; last 3 years for National 1979.

Source: Clark and Midanik, 1982.

A series of national studies conducted between 1971 and 1979 which examined trends in alcohol consumption (see table II-4) found little change in the level of drinking over that time. While an upward trend in heavier drinking by males was seen between 1971 and 1976, the 1979 national survey indicated that heavier drinking by males reverted to the 1971 and 1972 levels.

The prevalence of use of illicit drugs was monitored between 1972 and 1982 by a series of national household surveys. The 1982 national survey (Miller, Cisin, Gardner-Keaton, Harrell, Wirtz, Abelson, and Fishburne, 1983) indicated that 10.9 percent of all adults used marijuana within the month prior to the survey. This included 27.4 percent of young adults (ages 18 to 25) and 6.5 percent of older adults (see table II-5). The surveys indicated that current use of marijuana by youth and young adults peaked in 1979 and dropped significantly by 1982 (see table II-6). Data indicated no statistically significant change in the level of use by older adults.

The national surveys also assessed use of cocaine, heroin, and hallucinogens, as well as nonmedical use of various prescription drugs. Current heroin use for youth in 1982 was below one-half of 1 percent, and current use of each of the other drugs (use in past month) was between 1 and 2 percent. Current use of drugs by young adults was in the same range for everything except cocaine (6.8 percent) and stimulants (4.7 percent). The drop in cocaine use by young adults from the 9.3 percent observed in 1979 was statistically significant. Abuse of other drugs by older adults in the month prior to the survey was less than 1 percent for everything except cocaine (1.1 percent).

Studies of alcohol abuse have focused on both lifetime patterns and current levels of consumption as well as impacts of alcohol consumption on social functioning. Alcohol abuse has been increasingly defined in terms of its consequences. Close attention has been paid to undesirable impacts on family, friends, strangers, and those in the household and at work, as well as in social situations.

The diagnostic criteria used in making prevalence estimates for drug abuse differed significantly from those used for alcohol abuse and other mental illnesses estimates. In the field of drug abuse, primary emphasis has been placed on lifetime patterns and recency of use of illicit sub-

Table II-4

Trends in Alcohol Consumption, 1971-1979, by Type of Drinker and Sex (in Percentages)

	Harris	Harris	Harris		Harris	ORC	RAC	National
	1971	1972	Spring 1973	Fall 1973	1974	1975	1976	1979
Type of Drinker								
Abstainer	36	36	34	37	36	36	33	33
Lighter	34	32	29	30	28	31	38	34
Moderate	20	23	23	21	28	21	19	24
Heavier	10	10	14	11	11	12	10	9
(N)*	(2,195)	(1,544)	(1,583)	(1,603)	(1,578)	(1,071)	(2,510)	(1,772)
Males								
Abstainer	30	28	25	26	24	27	25	25
Lighter	29	29	24	29	24	27	33	29
Moderate	26	28	29	26	34	26	24	31
Heavier	15	15	22	19	18	20	18**	14
Females								
Abstainer	42	44	42	47	42	45	39	40
Lighter	40	34	35	32	32	35	44	38
Moderate	13	18	17	17	21	15	15	18
Heavier	5	4	6	4	5	4	3	4

Note. Percentage are weighted figures and may not total to 100% due to rounding.

*N's presented are the same as in the Third Special Report to the U.S. Congress on Alcohol and Health (Noble, 1978); however, actual N's used in this analysis varied slightly.

**Statistically significant linear trend ($p<.05$), indicating an increase (excluding 1979 survey).

Source: Clark and Midanik, 1982.

Table II-5

Prevalence of Drug Abuse by Type of Drug and Age Groups, 1982
(in Percentages)

	Youth (age 12-17)		Young Adults (age 18-25)		Older Adults (age 26+)	
	Used Ever	Month	Used Ever	Month	Used Ever	Month
Marijuana	26.7	11.5	64.1	27.4	23.0	6.6
Hallucinogens	' 2	1.4	21.1	1.7	6.4	*
Cocaine	6.5	1.6	28.3	6.8	8.5	1.2
Heroin	*	*	1.2	*	1.1	*
Nonmedical use of:						
Stimulants	6.7	2.6	18.0	4.7	6.2	.6
Sedatives	5.8	1.3	18.7	2.6	4.8	*
Tranquilizers	4.9	.9	15.1	1.6	3.6	*
Analgesics	4.2	.7	12.1	1.0	3.2	*
Any Nonmedical Use	10.3	3.8	28.4	7.0	8.8	1.2
Alcohol	65.2	26.9	94.6	67.9	88.2	56.7
Cigarettes	59.5	14.7	76.9	39.5	78.7	34.6

*Less than 5%.

Source: Miller et al., 1983.

Table II-6

Use of Drugs in Past Month, 1972-1982, by Young Adults

Young Adults: Age 18-25	1972 (772)	1974 (849)	1976 (882)	1977 (1500)	1979 (2400)	1982 (1283)	Change '79-'82**
Marijuana	27.8	25.2	25.0	27.4	35.4	27.4	SSS
Hallucinogens	†	2.5	1.1	2.0	4.4	1.7	SSS
Cocaine	†	3.1	2.0	3.7	9.3	6.8	S
Heroin	†	*	*	*	*	*	--
Nonmedical Use of:							
Stimulants	x	3.7	4.7	2.5·	3.5	4.7	§
Sedatives	x	1.6	2.3	2.8·	2.8	2.6	§
Tranquilizers	x	1.2	2.6	2.4·	2.1	1.6	§
Analgesics	x	x	*	x	1.0	1.0	§
Any Nonmedical Use	xx	xx	xx	xx	6.2	7.0	§
Alcohol	x	69.3	69.0	70.0	75.9#	67.9#	SSS
Cigarettes	x	38.8	39.3	37.2	--	39.5	--
(Alternate Definition-Cigarettes)··					(42.6)	(38.0)	(S)

*Less than .5%

xNot asked.

xxSince questions on use of analgesics were not asked in surveys prior to 1979, the nonmedical use of any psychotherapeutic (including analgesics) could not be reported for these earlier years.

†Not tabulated

·1977 estimates based on split sample: N=750.

··In 1979, recency of cigarette use was asked only of those who had smoked at least five packs during their lifetime. In all other years, no such restriction was applied. For 1982, this version was calculated separately.

#In both 1979 and 1982, private answer sheets were used for alcohol questions; in earlier years respondents answered these questions aloud.

**Significance levels: SSS, .001; SS, .01; S, 05; $, .10; NS, not significant; §, significance test not performed ('79-82 procedures not comparable).

Source: Miller et al., 1983.

stances or nonmedical use of various prescription drugs. While undesirable impacts of drug abuse are hypothesized and, in fact, observed, they have not been rigorously examined in a manner analogous to that for alcohol abuse. The inclusion of drug abuse and drug dependency in DSM-III and, in the ECA projects, the Diagnostic Interview Schedule may lead to notable developments in the drug abuse field. As the DSM-III is refined, and eventually implemented through community-wide and nationwide surveys, it can be expected that an increasingly better understanding of the extent and manifestations of drug abuse will be gained.

III. COST ESTIMATES

A. Introduction

The estimates produced in this cost study have been generated using the general methodology described in the 1981 RTI study. The report of the previous study documents the rationale behind individual cost components, the equations used to generate the estimates, and the sources of data that were utilized. The reader is encouraged to review this methodology. Wherever possible, this study has used the same or similar data sources. New data sources and minor modifications to the 1981 methodology are described below.

Significant changes and extensions in cost estimation have been made for several components. The basic estimates and computations for these components are presented in this section. More detailed discussion and documentation appears in the appendix on fetal alcohol syndrome, reduced productivity due to alcohol abuse and drug abuse, the relationship of homicide and other violent crimes to drug abuse, mental illness and crime, and the cost of administering social welfare benefits to ADM victims.

Total cost estimates are presented in table III-1. A guide to these data and tables used to derive the final estimates are provided in table III-2. For a specific cost component of interest in table III-1, identify the component name and the ADM disorder, and refer to table III-2 to obtain the reference table numbers.

B. Core Direct Costs--Treatment

Inpatient and outpatient services are available from many types of institutions and health treatment and service providers. A first step in the prior study was to identify a consistent and reasonably exhaustive set of treatment settings that provide health care. After adjusting the data using information from NIMH, a list of settings consistent with data maintained by the Health Care Financing Administration (HCFA) of DHHS, NIAAA, NIDA, and NIMH was established.

For each setting, the study team accumulated information on the total value of goods and services provided. These values were obtained from HCFA, NIDA, NIAAA, and NIMH (see table III-3 for sources). For the same settings, data on service utilization by individuals with particular diseases and illnesses (ICD-9 diagnostic codes) were also obtained (see table III-3

Table III-1

Costs to Society of Alcohol Abuse, Drug Abuse
and Mental Illness, 1980
($ in millions)

	Alcohol Abuse	Drug Abuse	Mental Illness	Total
Core Costs	$79,607	$29,451	$52,418	$161,476
Direct				
Treatment	9,487	1,200	20,961	31,647
Support	984	243	2,597	3,823
Indirect				
Mortality	14,456	1,980	7,196	23,632
Morbidity[b]	54,680	26,028	21,664	102,372
Reduced productivity	(50,575)	(25,716)	(3,122)	(79,413)
Lost employment	(4,105)	(312)	(18,542)	(22,959)
Other Related Costs	9,919	17,485	1,818	29,222
Direct				
Motor vehicle crashes (property loss)	2,185	a	-	2,185
Crime[b]	2,347	5,910	870	9,127
Public	(2,062)	(4,454)	(635)	(7,151)
Private	(261)	(1,345)	(235)	(1,841)
Property loss/damage	(24)	(111)	(-)	(135)
Social welfare program	38	2	201	241
Other	2,912	537	659	4,108
Indirect				
Victims of Crime	172	845	-	1,017
Crime careers	-	8,725	-	8,725
Incarceration	1,801	1,466	88	3,356
Motor vehicle crashes (time loss)	464	a	-	464
Total	$89,526[c]	$46,936[c]	$54,236[c]	$190,698

Totals may not add due to rounding.

[a] Although costs are hypothesized to occur in this category, sufficient data are not available to develop a reliable estimate.

[b] Components are indicated in parentheses.

[c] The total costs to society for each of the three ADM disorders are not comparable, since the completeness of data available for each cost category varied significantly. For example, the estimate of reduced productivity is relatively complete for alcohol abuse, only partially complete for drug abuse, and incomplete for mental illness.

Source: Research Triangle Institute.

Table III-2

Table Reference List for Chapter 3

	Alcohol Abuse	Drug Abuse	Mental Illness
Core Costs			
Treatment	3,4,5	3,4,5	3,4,5
Support	6	6	6
Mortality	7,10,11	8,10,12	9,10,13
Morbidity[b]			
Reduced productivity	14,15,16,17,18	14,15,22,23	14,15,25
Lost employment	19,20,21,37	24,37	26,27
Other Related Costs			
Motor vehicle crashes (property loss)	28	-	-
Crime[b]			
Public	29	30	31
Private	32	32	32
Property loss/damage	33	33	-
Social welfare program	34,35	34,35	34,35
Other	36	36	36
Victims of crime	37	37	-
Crime careers	-	38	-
Incarceration	39	39	39

Table III-3

Sources of Cost and Diagnostic Data Used to Estimate Costs for ADM Settings, 1980

Setting	Data Sources Costs	 Diagnoses
ADM facilities		
Hospital-based		
State and county psychiatric hospitals	NIMH^{1}	NIMH^{10}
Private psychiatric hospitals	NIMH^{1}	NIMH^{10}
VA neuropsychiatric hospitals	NIMH^{1}	NIMH^{10}
Non-Federal general hospitals with separate psychiatric units	NIMH^{1}	NIMH^{10}
Other ADM facilities and services		
Federally funded CMHCs	NIMH^{1}	NIMH^{11}
Residential treatment centers for children	NIMH^{1}	NIMH^{1}
Halfway houses		
Freestanding facilities	NIMH^{1}, NIDA^{2}, NIAAA^{3} .	NIDA^{2}, NIAAA^{3}, NIMH^{10}
Other facilities	NIMH^{1}, NIDA^{2}, NIAAA^{3}	NIMH^{1}, NIDA^{2}, NIAAA^{3}
ADM units in correctional facilities	NIDA^{2}, NIAAA^{3}	NIDA^{2}, NIAAA^{3}
Private practice psychiatrists	NHE^{4}	NCHS^{12}
Private practice psychologists	ADA^{5}	NCHS^{12}
General health facilities		
Hospital-based		
Non-Federal community hospitals (excluding psychiatric units)	NHE^{4}	NCHS^{13}
VA general hospitals and other facilities	Statistical Abstract^{6} Special data analysis^{7}	Special data analysis^{7}
Other Federal facilities	Federal agencies^{8} Statistical Abstract^{6}	Federal agencies^{14}

Table III-3 (continued)

Setting	Data Sources	
	Costs	Diagnoses
Other general health facilities and services		
Nursing homes	NHD[4]	NCHS[15]
Private practice physicians	NHE[4]	NCHS[12]
Dentists	NHE[4]	Estimated[16]
Other health professionals	NHE[4]	Estimated[16]
Drugs and drug sundries	NHE[4]	Estimated[16]
Other health services	NHE[4]	Estimated[16]
Volunteer services	ACTION[9]	Estimated[16]

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[1] U.S. DHHS, NIMH (1983a).

[2] U.S. DHHS, NIDA, special data analysis of 1980 NDATUS files.

[3] U.S. DHHS, NIAAA, special data analysis of 1980 NDATUS files.

[4] Gibson and Waldo (1981).

[5] Vandenbos, Stapp, and Kilburg (1981).

[6] U.S., DOC, BOC, Statistical Abstract (1982).

[7] Special data analysis, VA Department of Medicine and Surgery.

[8] Letters from: Department of the Air Force, Navy, Army, Public Health Service, and Indian Health Service.

[9] ACTION (1975; 1976).

[10] U.S. DHHS, NIMH (1983b).

[11] U.S. DHHS, NIMH (1981).

[12] U.S. DHHS, NCHS, unpublished 1980 Ambulatory Care Survey data.

[13] U.S. DHHS, NCHS, unpublished 1980 Hospital Discharge Survey data.

Table III-3 (continued)

[14] Unpublished reports from:

Public Health Service - *Primary Diagnosis by Sex and Age Group by Average Length of Stay for Single and Multiple Diagnosis, October 1976 through September 1977*.

Indian Health Service - *Inpatient Report 28 - Number of Discharges and Hospital Days by External Cause of Current Injury, by Age Group*. *Direct Inpatient on Request Report No. 21 - Hospital Discharges, Days and Average Length of Stay by Admission Diagnosis, Sex and Age Groups*.

Department of the Army - *Disposition, Bed Days, and Length of Stay by Selected Diagnosis, Patients in U.S. Army Hospitals Worldwide, CY 1977*.

Department of the Air Force - *Total Inpatient Discharges and Total Bed Days at Air Force Medical Treatment Facilities by Diagnosis Class and for Selected Diagnosis Within Selected Classes, Worldwide: FY 1977*.

Department of the Navy - *Incidence, Patients Treated in Navy Medical Treatment Facilities by Patient Category, 1976*.

[15] U.S. DHEW, NCHS (1979).

[16] Estimates based upon distribution of ADM illness costs in VA general hospitals, community hospitals, and for physician services.

for sources). Total expenditures within a given setting were allocated to alcohol abuse, drug abuse, and mental illness on the basis of service utilization data. This approach involved identifying specific diseases and illnesses that are related to alcohol, drug abuse and mental illness (see table III-4) and allocating costs based on the portions of the illnesses or diseases that are ADM-related. The causal relationship factors used here are identical to those in the 1981 study, except for the addition of alcoholic cardiomyopathy. For this study, HCFA provided 1980 expenditure data for most health care settings. For a number of ADM speciality settings (community mental health centers and various freestanding facilities), expenditure data were obtained from NIMH for 1979, the most recent year for which data had been collected. The 1979 estimates were inflated to 1980 values using the health care component index of the Consumer Price Index.

Sources of current utilization data included the Hospital Discharge Survey data for general hospitals, the Ambulatory Care Survey data for private practice physician visits, and inpatient data for all hospitals run by the Veterans Administration. Health care providers covered by these three data sources accounted for nearly 60 percent of all health care services utilized in the United States in 1977. Where 1980 data were unavailable, the 1977 utilization data for these settings were used.

Costs estimated using national expenditure and utilization data are presented in table III-5. The total costs for alcohol abuse, drug abuse, and mental illness, respectively, were $8.8 billion, $1.2 billion, and $21.0 billion, respectively, for a total of $30.9 billion. ADM accounted for approximately 14 percent of the $223.8 billion spent on personal health care for all illnesses in 1980.

Nearly half of total expenditures for mental illness ($21.0 billion) were for specialized services ($10.0 billion) such as psychiatric hospitals, CMHC's, and psychiatrists and psychologists. In contrast, 38 percent of expenditures for drug abuse and 12 percent for alcohol abuse were in specialized settings.

A major modification was made to this cost component with the introduction of cost estimates for the fetal alcohol syndrome (FAS). FAS may be described as a "set" of serious birth defects including mental impairment,

Table III-4

Diagnostic Categories Used to Estimate ADM Treatment Costs

A. ALCOHOL ABUSE

1. Alcohol Abuse-Specific Illnesses

ICDA-8 Code	ICD-9-CM Code	Diagnosis	Percent Associated with Alcohol Abuse
291	291	Alcohol psychosis	100
303	303	Alcohol dependence syndrome	100
	305.0	Alcohol abuse	100
571.0	571.0	Alcoholic fatty liver	100
571.0	571.1	Acute alcoholic hepatitis	100
571.0	571.2	Alcoholic cirrhosis of liver	100
571.0	571.3	Alcholic liver damage, unspecified	100
980.0	980.0	Toxic effects of ethyl alcohol	
	425.5	Alcoholic cardiomyopathy	100

2. Alcohol Abuse-Related Illnesses

Cancer

140-149	140-149	Malignant neoplasm of lip, oral cavity and pharynx	2 to 43
150	150	Malignant neoplasm of esophagus	28.8 to 80
151	151	Malignant neoplasm of stomach	.3 to 20
153	153	Malignant neoplasm of colon	.3
154	154	Malignant neoplasm of rectum, rectosigmoid junction, and anus	.3
155.0	155.0	Liver, primary	12.6
157	157	Malignant neoplasm of pancreas	1.2 to 75

Mental Disorders

296	296	Affective psychoses	2.8 to 42
300	300	Neurotic disorders	5.6

Infectious Diseases

011	011	Pulmonary tuberculosis	10.2 to 70
012	012	Other respiratory tuberculosis	10.2 to 70
480-486	480-486	Pneumonia	1.1 to 4

Table III-4 (continued)

ICDA-8 Code	ICD-9-CM Code	Diagnosis	Percent Associated with Alcohol Abuse
Gastrointestinal Tract			
456	456	Varicose veins of other sites	26.7
531	531	Gastric ulcers	1.2
536	536	Disorders of function of stomach	.8
532	532	Duodenal ulcer	.8 to 6.7
533	533	Peptic ulcer, site unspecified	.6 to 24.4
534	534	Gastrojejunal ulcer	1.8
535	535	Gastritis and duodenitis	2.9
577	577	Diseases of pancreas	11.6 to 58
Liver Disease			
571	571	Chronic liver disease and cirrhosis	64.75
Nervous Disease			
345	345	Epilepsy	3.3
Heart			
427.4	427.3	Cardiac arrhythmias	1.3 to 3.7
Endocrine System			
240-246	240-246	Disease of thyroid gland	.8 to 34.8
250-258	250-259	Diseases of other endocrine glands	.8 to 34.8
Nutritional Deficiency			
260-269	260-269	Nutritional deficiencies	4.3 to 60
274	274	Gout	1.7 to 25.3
275	273	Other and unspecified metabolic diseases	1.4
		3. Alcohol Abuse-Related Trauma	
800-999	800-999	Injury and poisoning	10

Table III-4 (continued)

ICDA-8 Code	ICD-9-CM Code	Diagnosis
B. DRUG ABUSE		
304	304	Drug dependence
	292	Drug psychosis
	305	Nondependent abuse of drugs
960-977	960-977	Poisonings of drugs, medicinal and biological substances
C. MENTAL ILLNESS		
290	290	Senile and presenile organic psychotic disorders
	293	Transient organic psychotic conditions
	294	Other organic psychotic conditions
295	295	Schizophrenic disorders
296	296	Affective psychoses
297	297	Paranoid states
298	298	Other nonorganic psychoses
299	299	Psychoses with origin specific to childhood
300	300	Neurotic disorders
301	301	Personality disorders
302	302	Sexual deviations and disorders
305	306	Physiological malfunction arising from mental disorders
306	307	Special symptoms or syndromes, nec
	308	Acute reaction to stress
307	309	Adjustment reaction
	310	Specific nonpsychotic mental disorders due to organic brain damage
	311	Depressive disorders, nec
	312	Disturbance of conduct, nec
	313	Disturbance of emotion specific to childhood and adolescence
	314	Hyperkinetic syndrome of childhood
	315	Specific delays in development
	316	Psychic factors associated with diseases classified elsewhere
794	794	Senility without mention of psychosis

Source: Research Triangle Institute.

Table III-5

ADM Costs by Setting, 1980
($ in millions)

Settings	Costs							
	All Alcohol Abuse Illnesses	Alcohol Abuse Specific Illnesses	Alcohol Abuse Related Trauma	Alcohol Abuse Related Illness	Drug Abuse	Mental Illness	All ADM	All Illnesses
ADM facilities	$1,058	$1,011	$ a	$ 47	$ 452	$10,070	$11,531	$ 12,375
Hospital-based	341	341			85	5,665	6,090	6,549
State and county psychiatric hospitals	217	217			54	3,605	3,876	4,167
Private psychiatric hospitals	43	43			11	713	767	824
VA neurosychiatric hospitals	33	33			8	543	584	620
Non-Federal general hospitals with separate psychiatric units	48	48			12	804	864	929
Other ADM facilities and services	717	670	a	47	367	4,357	5,440	5,827
Federally funded CMHCs	181	181			50	997	1,228	1,401
Residential treatment centers for children	0	0			0	484	484	484
Freestanding facilities	379	379			265	565	1,208	1,254
Other facilities	49	49			33	179	261	276
ADM units in correctional facilities	2	2			8	-	10	10
Private practice psychiatrists	58	11	a	47	6	1,150	1,213	1,286
Private practice psychologists	49	49			5	982	1,036	1,036
General health facilities	7,730	3,442	1,788	2,500	747	10,940	19,417	211,390
Hospital-based	4,800	2,386	882	1,532	527	5,088	10,415	93,046
Non-Federal community hospitals (excluding psychiatric units)	3,979	1,795	822	1,362	421	3,933	8,332	83,500
VA general hospitals and other facilities	544	435	18	91	46	861	1,451	5,313
Other Federal facilities[b]	278	157	43	78	60	294	631	4,233
Other general health facilities and services	2,930	1,056	906	968	221	5,852	9,002	118,350
Nursing homes	167	105	62	-	-	2,783	2,951	20,700
Private practice physicians	726	61	373	291	28	870	1,624	45,514
Dentists	621	271	144	206	59	670	1,350	15,900
Other health professionals	171	74	39	57	16	184	371	4,364
Drugs and drug sundries	750	327	173	249	71	810	1,631	19,200
Other health services	359	157	83	119	34	388	781	9,195
Volunteer services	136	59	31	45	13	146	295	3,472
Total	$8,787	$4,453	$1,788	$2,546	$1,200	$20,961	$30,947	$223,770

Note: Totals may not add due to rounding.

[a] Less than $.5 million.

[b] A small portion of these were in non-hospital-based facilities.

Source: Research Triangle Institute.

growth retardation, hearing problems, heart defects, and other abnormalities. The defects occur at varying rates within the syndrome, with no single combination of defects predominant.

The costs of FAS could not be estimated using the established methodology for several reasons. Children with birth defects may not be identified as FAS victims when they are treated and, therefore, are not identified in health treatment statistics. Each of the individual defects also may arise due to other causes. The syndrome was only recently identified by health researchers, and it is not widely understood in the profession. Although FAS has a rate of incidence close to those of Down's syndrome and spina bifida, it is still relatively infrequent.

Estimation of the majority of treatment costs for FAS was based on nationally representative data sources for health treatment expenditures and utilization. The methodological technique and results are presented in the appendix. The mid-range cost for FAS treatment in 1980 was $2.4 billion. The largest cost component was for the provision of institutional and day services for adult FAS victims with mental retardation. The findings are consistent with and additive to the other direct health treatment costs for alcohol abuse and have been included in tables III-1 and I-1.

C. Core Direct Costs--Support

The methodology for these costs has been reproduced from the prior study (table III-6).

D. Core Indirect Costs--Mortality

The methodology for these costs has been reproduced from the prior study. There are three crucial elements in deriving the mortality cost estimates: the identification of the diagnoses related to ADM deaths, the numbers of ADM deaths, and the value of these lost lives. A number of illnesses and diseases have been identified in publications by NIAAA, NIDA, and NIMH as being ADM-related (see tables III-7, III-8, and III-9). This study and the prior effort generally used factors that have been identified in published work. Alcoholic cardiomyopathy, a higher proportion of mortality due to "other" cirrhosis of the liver, and 10 percent of homicides (due to violence in the drug distribution network) were added in this study. The second element is the number of deaths by illness or disease type. Mortality data for 1980 were obtained from the National Center for Health Statistics, Vital Statistics Branch. The third element is the value placed on those lost lives.

Table III-6

Support Costs for ADM Health Care Provision, 1980
($ in millions)

Expenditure Category	Total Health Expenditures	Share of Total Health Services and Supplies Due to			Costs			
		Alcohol Abuse (percent)	Drug Abuse (percent)	Mental Illness (percent)	Alcohol Abuse	Drug Abuse	Mental Illness	All ADM Disorders
Research[a]	$ 5,400	0.70	2.10	6.30	$ 37.8	$113.4	$ 340.2	$ 491.4
Training & education[b]	7,600	3.93	0.54	9.40	298.0	40.7	710.7	1,049.0
Construction[b]	6,100	3.93	0.54	9.40	239.5	32.7	571.4	843.6
Health insurance administration[b]	10,400	3.93	0.54	9.40	408.4	55.8	974.2	1,438.0
Total	$29,500				$983.7	$242.5	$2,596.5	$3,822.8

[a] From the Report by the President's Commission on Mental Health (1978).

[b] Calculated by assuming that the ADM portions of total expenditures for this category are the same as the ADM portion for direct health care treatment.

Totals may not add due to rounding.

Source: Research Triangle Institute.

Table III-7

Causes of Mortality Related to Alcohol Abuse

Cause	Proportion of Mortalities Due to Alcohol Abuse (percent)[a]	Mortalities in 1980	ICDA-8 Code	ICD-9 Code
Direct causes - primary				
Alcoholic psychoses	100.0	454	291	291
Alcoholism	100.0	4,345	303	303
Cirrhosis, alcoholic	100.0	12,705	571.0	571.0-571.3
Accidental poisoning by alcohol	100.0	343	E860	E860
Cardiomyopathy, alcoholic	100.0	650	425	425.5
Direct causes - secondary				
Cirrhosis, other	64.3	16,170	571.8,571.9	571.4-571.9
Respiratory tuberculosis	25.0	1,634	011-012	011-012
Neoplasm				
Tongue, malignant	-	1,874	141	141
Mouth, floor malignant	-	509	144	144
Mouth, other, malignant	-	1,399	145	145
Pharynx, malignant, oro	-	1,127	146	146
Pharynx, malignant, naso	-	585	147	147
Pharynx, malignant, hypo	-	634	148	148
Pharynx, malignant, unspecified	-	1,501	149	149.0
Esophagus, malignant	-	7,985	150	150
Stomach, malignant	-	14,372	151	151
Rectum, malignant	-	7,435	154.1	154.1
Liver, malignant, primary	56.0	2,395	155.0	155.0
Liver, bile duct type[b]	-	455	155.1	155.1
Pancreas, malignant	-	19,640	157.9	157.9
Larynx, malignant	-	3,412	161	161
Liver, malignant, unspecified	-	-	197.8	197.7
Esophagus, benign	-	-	211.0	211.0
Liver, benign[b]	-	11	211.5	211.5
Rectum, benign	-	1	211.4	211.4
Pancreas, benign	-	9	211.6	211.6
Larynx, benign	-	1	212	212.1
Stomach, unspecified	-	2	230	230.2
Esophagus, unspecified	-	1	230.0	230.1
Liver, unspecified	-	-	230.5	230.8
Pancreas, unspecified	-	-	230.6	230.9
Rectum, unspecified	-	-	230.4	230.4
Larynx, unspecified	-	1	231	231.0
Tongue, unspecified[b]	-	4	239	230

Table III-7 (continued)

Cause	Proportion of Mortalities Due to Alcohol Abuse (percent)[a]	Mortalities in 1980	ICDA-8 Code	ICD-9 Code
Hypoglycemia, spontaneous	-	167	251	251.0, 251.2
Malabsorption[b]	-	11	261	265.1
Beriberi	-	2	261	265.0
Polyneuropathy[b]	-	11	261	265.1
Niacin deficiency[b]	-	4	262	265.2
Pellagra	-	4	262	265.2
Malabsorption	-	4	263.8	266.2
Cyanocobalamine deficiency[b]				
Folic acid deficiency[b]				
Other vitamin B deficiencies, unspecified	-	-	263.9	266.9
Wernicke's encephalopathy[b]	-	-	263.9	266.9
Ascorbic acid deficiency	-	4	264	267
Malabsorption syndrome, unspecified	-	-	269.1	-
Plasma protein abnormalities	-	375	275	273
Other and unspecified metabolic diseases	-	1,361	279	277
Folic acid deficiency anemia	-	14	281.2	281.2
Other acquired hemolytic anemias	-	25	283.9	283.9
Progressive muscular dystrophy	-	589	330.3	359.1
Other demyelinating diseases of central nervous system	-	182	341	341
Marchiafava's disease[b]	-	4	341	341.8
Other and unspecified diseases of peripheral nerve except autonomic	-	109	357.9	356.8, 356.9
Hypertension[b]	-	2,840	401	401
Diseases of capillaries	-	45	448	448
Inflammatory diseases of esophagus	-	248	530.1	530.1
Gastritis and duodenitis	-	695	535	535
Pancreatitis, acute	35.5	1,390	577.0	577.0
Pancreatitis, chronic	35.5	261	577.1	577.1
Rosacea	-	0	695.3	695.3
Chronic ulcer of skin, other[b]	-	70	707.9	707.8, 707.9
Other and unspecified disease of muscle, tendon, and fascia	-	72	733.9	728.8, 728.9

Table III-7 (continued)

Cause	Proportion of Mortalities Due to Alcohol Abuse (percent)[a]	Mortalities in 1980	ICDA-8 Code	ICD-9 Code
Myopathy, primary	-	72	733.9	728.8, 728.9
Myopathy, progressive	-	72	733.9	728.8, 728.9
Convulsions[b]	-	354	780.2	780.3
Keto-acidosis, alcoholic[b]	-	710	788.0	276.2
Senility without mention of psychosis	-	1,233	794	797
Indirect causes				
Motor vehicle crashes	37	52,979	E810-E823	E810-E825
Accidental falls	44.4	13,312	E880-E887	E880-E888
Accidents caused by fires and flames	25.9	5,865	E890-E899	E890-E899
Homicide	30.0	23,902	E960-E969	E960-E969
Other accidents	11.1	30,573	E830-E845, E910-E929	E800-E807 E830-E845 EE860-E879 E900-E949
Suicide	0	26,852		

[a] Because the proportion of mortalities due to alcohol abuse is unknown, some cells in this column lack a number giving a proportion. These causes are excluded from the analysis.

[b] This illness is a subcategory of the ICD-9 class for which mortality data are available. Therefore, the number of deaths due to this cause is less than the value listed.

Source: Keller (1971; 1974); Noble (1978); unpublished mortality data from the Vital Statistics files provided by NCHS (U.S. DHHS, NCHS, no date).

Table III-8

Causes of Mortality Related to Drug Abuse

Cause	Proportion of Mortalities Due to Drug Abuse (percent)^a	Mortalities in 1980	ICDA-8 Code	ICD-9-CM Code
Drug Dependence	100	629	304	304
Accidental Overdose of Psychoactive Drugs				
Heroin	100	322	E853.0	E850.0
Methadone	100	75	E853.0	E850.1
Other opiates and related narcotics	100	21	E853.0	E850.2
Other nonnarcotic analgesics	100	0		E850.7
Other specific analgesics and antipyretics	100	97	E858.9	E850.8
Unspecified analgesics and antipyretics	100	0	E853.9	E850.9
Barbiturates	100	154	E854.0	E851
Other sedatives and hypnotics	100	59	E854	E852
Tranquilizers	100	110	E855.1	E853
Other psychotropic agents (inc. antidepressants)	100	161	E855.2	E854
Other drugs acting on central and autonomic nervous system	100	159	E856	E855
Agricultural and horticultural chemical and pharmaceutical preparations other than plant foods and fertilizers	100	24	E865	E863
Accidental Overdose of Drugs and Medicaments				
Accidental poisoning by antibiotics	100	50	E850	E856
Accidental poisoning by other anti-infectives	100	7	E850	E857
Hormones and synthetic substitutes	100	20	E851	E858.0
Primarily systemic agents	100	12	E852	E858.1
Agents primarily affecting blood constituents	100	17	E852	E858.2
Salicylates	100	2	E853.1	E850.3
Aromatic analgesics, nec	100	2	E853.4	E850.4
Pyrazole derivatives	100	117	E853.5	E850.5
Antiheumatics	100	0		E850.6

Table III-8 (continued)

Cause	Proportion of Mortalities Due to Drug Abuse (percent)[a]	Mortalities in 1980	ICDA-8 Code	ICD-9-CM Code
Agents primarily affecting cardiovascular system	100	190	E857	E858.3
Agents primarily affecting gastrointestinal system	100	3	E858	E858.4
Water mineral and uric acid metabolism drugs	100	13	E859.3	E858.5
Agents primarily acting on the smooth and skeletal muscles and respiratory system	100	13	E859.4	E858.6
Agents primarily affecting skin and mucus membrane ophthalmological, otorhino-laryngological, and dental drugs	100	9	E859.5 E859.6 E859.7	E858.7
Other specified drugs	100	404	E859.8	E858.8
Unspecified drug	100		E859.9	E858.8
Injury Undetermined Whether Accidental or Purposely Inflicted				
Analgesics, antipyretics and antiheumatics	100	201	E980.1	E980.0
Barbiturates	100	65	E980.0	E980.1
Other sedatives and hypnotics	100	28		E980.2
Tranquilizers and other psychotropic agents	100	140	E980.2	E980.3
Other specified drugs and medicinal substances	100	174	E980.3	E980.4
Unspecified drug or medicinal substance	100	217	E980.3	E980.5
All other solid or liquid substances	100	507	E980	E980

Source: Unpublished mortality data from the Vital Statistics files provided by NCHS (U.S. DHHS, NCHS, no date).

Table III-9

Causes of Mortality Related to Mental Illness

Cause	Proportion of Mortalities due to Mental Illness (percent)[a]	Mortalities in 1980	ICDA-8 Code	ICD-9-CM Code
Psychoses				
Senile and presenile organic psychotic	100	1,713	290	290
Transient organic psychotic conditions	100	2		293
Other organic psychotic conditions	100	41		294
Schizophrenic disorders	100	165	295	295
Affective psychoses	100	48	296	296
Paranoid states	100	7	297	297
Other nonorganic psychoses	100	437	298	298
Psychoses with origin specific to childhood	100	1	299	299
Neuroses				
Neurotic disorders	100	66	300	300
Personality disorders	100	2	301	301
Sexual deviations and disorders	100	1	302	302
Physiological malfunction arising from mental disorders	100	25	305	306
Special symptoms or syndromes, nec	100	162	306	307
Acute reaction to stress	100	32		308
Adjustment reaction	100	2	307	309
Specific nonpsychotic mental disorders due to organic brain damage	100	4,170	794	310
Depressive disorders, nec	100	217		311
Disturbance of conduct, nec	100	0		312
Disturbance of emotion specific to childhood and adolescence	100	0		313
Hyperkinetic syndrome of childhood	100	0		314
Specific delays in development	100	4		315
Psychic factors associated with diseases classified elsewhere	100	0		316

Table III-9 (continued)

Cause	Proportion of Mortalities Due to Mental Illness (percent)[a]	Mortalities in 1980	ICDA-8 Code	ICD-9-CM Code
Suicide and self-inflicted poisoning by solid or liquid substances	100	216	E950.4 E950.9	E950.6 E950.9
Suicide and self-inflicted poisoning by: analgesics, antipyretics, and antiheumatics, barbiturates, other sedatives and hynotics	100	607	E950.1 E950.0	E950.1 E950.1 E950.2
Tranquilizers and other psychotropic agents	100	754	E950.2	E950.3
Other specified drugs and medicinal substances	100	562	E950.3	E950.4
Unspecified drug or medicinal substances	100	538	E950.3	E950.5
Suicide: (excluding drug overdoses): E950.1, E950.2, E950.3, E950.4, E950.5	100	24,391		E950- E959

Source: Unpublished mortality data from the Vital Statistics files provided by NCHS (U.S. DHHS, NCHS, no date).

This study and its predecessors used the human capital approach which sums the present discounted value of productivity over the expected remaining lifetime. In this approach, a value is assigned to production lost due to premature death. These factors were obtained from Dr. Thomas Hodgson of NCHS (see table III-10). The cost estimates are presented as tables III-11, III-12, and III-13, respectively, for alcohol, drug abuse, and mental illness.

E. Core Indirect Costs--Morbidity

1. Alcohol Abuse

Reduced Productivity

Alcohol abuse is recognized as a significant problem in the workplace. This component estimates the losses to society due to impaired productivity on the job of persons that may or may not be recognized by employers as problem drinkers. A detailed discussion of the issues and methodology is found in the appendix.

This component was estimated at $50.6 billion in 1980, a 114 percent increase over the 1977 estimates. As the result of an analysis reported in the appendix, the impact of problem drinking on productivity in the workforce is now estimated at 21 percent instead of the 14 percent figure used in the previous study. The overall prevalence rate has not changed: it is estimated to be 10 percent of the workforce (see table III-17 for the age/sex prevalence).

Several other changes were made. Wages, salaries, and employee benefits increased by 31 percent between 1977 and 1980, and the workforce grew by 8 percent. To be consistent with the prior estimates for alcohol- and mental illness-related disabilities, costs were imputed to all problem drinkers in the labor force (an extra 9 percent), not just to those who were employed.

Fundamental information used in these calculations is found in tables III-14 to III-18. Average expected productivity rates by age and sex are shown in table III-14, while the total age/sex distribution of the labor force is shown in table III-15. Basic rates of problem drinking in the adult U.S. population from the most recent national household survey (Clark and Midanik, 1982) are found in table III-16. These are presented for comparison to the prevalence rates developed in that study (table III-17).

Table III-10

Present Values of Expected Future Lifetime Earnings and Housekeeping Services According to Age, Sex, and Discount Rate, 1980

Age	Male			Female		
	10%	6%	4%	10%	6%	4%
0-1	$56,173	$200,992	$415,998	$51,194	$166,303	$330,065
1-4	68,085	222,067	438,242	62,002	183,597	347,443
5-9	95,842	264,604	479,294	87,229	218,641	379,771
10-14	140,028	321,232	529,007	127,380	265,301	418,954
15-19	195,970	382,235	576,855	171,579	308,166	448,842
20-24	247,482	429,152	604,379	198,450	325,736	448,982
25-29	279,025	446,490	597,040	201,908	314,918	418,703
30-34	288,553	434,295	557,084	191,522	288,221	372,595
35-39	278,238	397,573	492,083	176,550	256,285	322,318
40-44	253,340	344,695	412,732	158,732	221,311	270,522
45-49	217,538	281,249	325,967	136,295	182,338	216,805
50-54	170,539	209,546	235,464	109,535	140,696	163,014
55-59	113,364	132,720	145,110	79,530	98,510	111,643
60-64	54,830	62,538	67,446	49,785	60,224	67,282
65-69	20,535	23,810	25,887	27,906	33,453	37,104
70-74	10,271	11,750	12,666	15,965	18,772	20,557
75-79	5,074	5,719	6,108	8,855	10,159	10,952
80-84	2,578	2,847	3,002	3,722	4,115	4,340
85+	852	892	914	1,116	1,169	1,197

Source: Unpublished data from Dr. Thomas Hodgson, NCHS (1984).

Table III-11
Economic Cost of Mortalities Due to Alcohol Abuse - for Various Discount Rates, 1980
($ in millions)

Cause of Death - Primary Diagnosis	Mortalities in 1980			Discount Rates		
	Total	Attributed to Alcohol Abuse				
		Percent	Number	10%	6%	4%
Direct Primary Causes	18,497	100	18,497	$2,325	$3,076	$3,645
Alcohol psychosis	454	100	454	60	80	96
Alcoholism	4,345	100	4,345	564	748	888
Alcohol cirrhosis of liver	12,705	100	12,705	1,555	2,047	2,418
Alcohol poisoning	343	100	343	59	85	107
Alcoholic cardiomyopathy	650	100	650	87	115	136
Direct Secondary Causes	92,218		13,086	1,183	1,563	1,656
Cirrhosis of liver-other	16,170	64.3	10,397	1,002	1,321	1,565
Malignant primary liver neoplasm	2,395	56	1,341	78	104	126
Other malignant neoplasms of gastrointestinal tract	60,958	a	-	-	-	-
Pancreatitis	1,651	35.5	939	79	107	129
Respiratory tuberculosis	1,634	25	409	24	31	36
Other associated diseases	9,410	a		-	-	-
Indirect Causes	153,483		37,597	6,067	9,817	13,410
Motor vehicle crashes	52,979	37	19,602	3,596	5,919	8,162
Falls	13,312	44.4	5,911	317	462	594
Fires	5,865	25.9	1,519	169	287	413
Other[b]	30,573	11.1	3,394	485	789	1,090
Homicide	23,902	30	7,171	1,500	2,358	3,148
Suicide	26,852	0	-	0	-	0
Total	264,198		$69,180	$9,575	$14,456	$18,911

[a] Values of causal factors unknown and are assumed to be zero for this study.

[b] Includes all accidents not listed above but excludes accidents incurred in medical and surgical procedures and psychoactive drugs.

Totals may not add due to rounding.

Source: Research Triangle Institute; unpublished mortality data from the Vital Statistics files provided by NCHS (U.S. DHHS, NCHS, no date).

Table III-12

Economic Cost of Mortalities Due to Drug Abuse - for Various Discount Rates, 1980 ($ in millions)

Cause of Death - Primary Diagnosis	Mortalities in 1980	Attributed to Drug Abuse		Discount Rates		
	Total	Percent	Number	10%	6%	4%
Drug dependence	629	100	629	$157	$242	$317
Accidental overdose of psychoactive drugs	1,182	100	1,182	239	371	490
Accidental overdose of other drugs and medicaments	859	100	859	129	200	265
Suicidal overdose of drugs and medicaments	2,461	0	0	0	0	0
Overdose of drugs and medicaments, reason undetermined	1,332	100	1,332	251	381	497
Homicide	23,902	10	2,390	500	786	1,049
Total	30,365		6,392	$1,276	$1,980	$2,618

Totals may not add due to rounding.

Source: Research Triangle Institute; unpublished mortality data from the Vital Statistics file provided by NCHS (U.S. DHHS, NCHS, no date).

Table III-13

Economic Cost of Mortalities Due to Mental Illness - for Various Discount Rates, 1980 ($ in millions)

Cause of Death - Primary Diagnosis	All Mortalities	Mortalities Attributed to Mental Illness		Discount Rates		
		Percent	Number	10%	6%	4%
Psychoses and neuroses[a]	7,095	100	7,095	$108	$141	$169
Alcoholic psychoses and alcoholism	454	0	0	0	0	0
Drug dependence	629	0	0	0	0	0
Suicide (excluding drug overdoses)	24,391	100	24,391	4,242	6,460	8,441
Suicidal overdose of drugs or medicaments	2,461	100	2,461	403	595	763
Homicide	23,902	0	0	0	0	0
Total	58,932		33,947	$4,752	$7,196	$9,373

Totals may not add due to rounding.

[a] Excluding alcohol and drug abuse-related deaths.

Source: Research Triangle Institute; unpublished mortality data from the Vital Statistics file provided by NCHS (U.S. DHHS, NCHS, no date).

Table III-14

Values of Market and Household Productivity
by Age and Sex Cohort, 1980

	Males		Females		
Age	Full-time Earnings with Wage Supplement	Imputed Household Value	Full-time Earnings with Wage Supplement	Employed Household Value	Nonemployed Household Value
15-19	$10,572	$ 70	$ 8,225	$ 89	$ 9,902
20-24	15,529	807	11,712	1,959	11,113
25-29	21,293	1,685	15,307	5,657	12,233
30-34	25,243	1,925	15,161	6,745	11,921
35-39	27,221	1,860	14,886	7,070	11,429
40-44	28,733	1,863	14,994	6,658	10,697
45-49	29,121	1,825	15,054	6,253	10,011
50-54	28,074	1,817	15,219	5,840	9,369
55-59	27,173	1,633	14,460	5,430	8,768
60-64	25,030	1,593	14,373	4,189	6,416

Sources: Paringer and Berk (1977); U.S. DOL, BLS, Monthly Labor Review (September, 1983).

Table III-15

U.S. Labor Force and Employment
by Age and Sex, 1980
(number in thousands)

	Males			Females		
Age	Labor Force	Unemployed	Employed	Labor Force	Unemployed	Employed
16-17	2,069	419	1,649	1,740	340	1,400
18-19	3,121	477	2,644	2,618	404	2,214
20-24	9,022	1,033	7,989	7,170	733	6,437
25-34	16,943	1,094	15,849	11,890	852	11,038
35-44	11,901	474	11,426	8,605	460	8,145
45-54	9,989	361	9,627	6,974	316	6,658
55-64	7,165	241	6,925	4,592	150	4,441
65+	1,877	58	1,819	1,444	36	1,108
Total	62,087	4,157	57,928	44,733	3,291	41,441

Source: U.S. DOL, BLS, Employment and Earnings (1981).

Table III-16

Proportions of Age-Sex Cohorts with Problems Due to Alcohol Abuse

Age	Proportion with Social Consequences		Proportion with Loss of Control or Dependence	
	Males	Females	Males	Females
18-20	15	5	35	16
21-25	13	6	25	13
26-30	10	3	25	7
31-40	8	5	16	8
41-50	2	4	8	5
51-60	3	1	5	4
61-70	5	0	6	0
70+	4	0	2	0
Total	7	3	15	6

Source: Research Triangle Institute; Clark and Midanik, 1982.

Table III-17

Proportions of Age-Sex Cohorts with
Productivity Reductions Due to Alcohol Abuse

	Proportion with One or More Problem Drinking Symptoms	
Age	Males	Females
18-19	16.4	5.7
20-24	21.0	15.4
25-34	18.5	4.5
35-44	11.9	8.2
45-54	13.3	5.6
55-64	6.0	0.8

Source: Research Triangle Institute; analysis of 1979 national household survey on alcohol abuse (Clark and Midanik, 1982).

The cost estimates of reduced productivity by age and sex are in table III-18. The number of persons in the workforce in each age/sex group was multiplied by the problem drinker prevalence rate for that group to indicate the number of problem drinkers. That number was then multiplied by the productivity values of III-14 and the rate of impairment (21 percent) to yield the value of their reduced productivity. The values in table III-18 for age groups 18 to 34 are reduced by 25 percent to adjust for the occurrence of alcohol abuse and drug abuse in the same people in this age range.

Another element was added to this cost component. The productivity of adult FAS victims is impaired due to their mental deficiencies. Impairments for minimal brain dysfunction, and mild and moderate mental retardation were assumed to be 10, 25 and 50 percent, respectively. The 219,000 non-institutionalized FAS victims had costs of $749 million. See the appendix for discussion of these estimates.

Lost Employment

There are four elements of this component: persons who experience acute problems and losses of productive time due to injuries from motor vehicle accidents, work place accidents, home accidents, violent crimes, or other kinds of trauma (table III-19); persons with chronic physical impairments which either prevent them from working or impair their ability to work (table III-20); persons out of the labor force receiving inpatient treatment (table III-21); and adult FAS victims severely/profoundly mentally retarded.

Acute short-term loss of employment due to trauma was estimated at $938 million in 1980, a 72 percent increase over the 1977 estimate of $545 million. The growth is due primarily to increases in wages and salaries in the labor force; however, the estimate also increased by an additional 30 percent due to better data from the U.S. DOJ, BJS (1984) on work loss due to violent crimes (see table III-37).

Lost employment due to physical impairments resulted in estimated costs of $2.2 billion in 1980 compared to $1.6 billion in 1977. This is an increase of about 37.5 percent, representing the combined effects of increases in wages and salaries and labor force growth over the three-year period.

Table III-18

Reduced Productivity in 1980: Alcohol Abuse Number of Problem Drinkers and Value of Reduced Productivity, by Age and Sex Cohort (Number in thousands, $ in millions)

			Females				
	Males		Employed		Housewives	Total	
Age	Number	Value	Number	Value	Value	Number	Value
18-19	699	$ 858	240	$ 195	$ 42	939	$1,096
20-24	2,178	4,875	1,596	2,378	554	3,773	7,806
25-34	3,285	12,338	817	1,804	476	4,102	14,619
35-44	1,482	8,852	1,078	3,232	783	2,560	12,868
45-54	1,456	8,487	652	1,737	470	2,108	10,694
55-64	594	2,525	89	150	69	683	2,743
Total	9,694	$37,935	4,471	$9,496	$2,394	14,165	$49,826

Totals may not add due to rounding.

Source: Research Triangle Institute; special analysis of the 1979 national household survey on alcohol abuse (Clark and Midanik, 1982).

Table III-19

Lost Employment in 1980: Victims of Alcohol-Related Trauma (Accidents and Violence)
(Number in thousands, $ in millions)

	Person Years	Cost
Motor vehicle accidents	8.2	$147.7
Work place accidents	9.9	178.7
Home accidents	9.1	157.2
Other accidents	14.5	256.8
Violent crimes	11.2	198.0[a]
Total	52.9	$938.4

Source: Research Triangle Institute; U.S. DHEW, NCHS; U.S. DOJ, LEAA, 1979c.

[a] Unpublished data from the U.S. DOJ, BJS.

Table III-20

Lost Employment in 1980: Persons with
Physical Impairments Due to Alcohol Abuse
(Number in thousands, $ in millions)

	Complete		Partial		Total	
	Number	Value	Number	Value	Number	Value
Paralysis,	5.1	$ 105.2	5.0	$ 14.4	10.1	$ 119.6
Absence of major extremities	0.9	18.9	2.4	7.7	3.3	26.6
Orthopedic impairments (except paralysis) of back or spine	23.3	465.2	92.8	263.9	116.1	729.1
Orthopedic impairments (except paralysis or absence) of upper extremity or shoulder	6.4	178.4	29.3	82.7	35.7	261.1
Orthopedic impairments (except paralysis or absence) of lower extremity or hip	18.4	372.4	40.2	116.1	58.6	488.5
Orthopedic impairments (except paralysis or absence), other, multiple, and ill-defined of limbs, back or trunk, not elsewhere classified	22.0	458.8	39.3	115.5	61.3	574.3
Total	76.1	$1,598.9	209.0	$600.3	285.1	$2,199.2

Source: Research Triangle Institute.

Table III-21

Value of Lost Employment in 1980:
Alcohol Abusers in Residential Treatment
($ in millions)

Age	Males		Females		Total	
	Number	Value	Number	Value	Number	Value
18-19	1,887	$ 15	542	$ 3	2,429	$ 18
20-24	4,330	62	1,244	15	5,575	76
25-34	8,138	195	2,338	42	10,475	236
35-44	8,138	232	2,338	41	10,475	273
45-54	4,964	138	1,426	23	6,391	161
55-64	4,038	84	1,160	14	5,199	98
65+	728	0	209	0	937	0
Total	32,222	$725	9,259	$138	41,481	$863

Totals may not add due to rounding.

Source: Research Triangle Institute; NDATUS; verbal communication with Pat Reed of NIAAA.

Table III-22

Labor Force Age-Sex Cohorts with Productivity Reductions Due to Drug Abuse

Age	Proportion with Daily Use (Ever) of Marijuana	
	Males	Females
18-19	18.9	14.3
20-24	21.8	11.0
25-34	12.5	4.5
35-44	0.0	0.0
45-54	0.0	0.0
55-64	0.0	0.0

Source: Research Triangle Institute: analysis of 1982 National Household Survey (Miller et al., 1983).

Table III-23

Reduced Productivity in 1980: Drug Abuse
Number of Problem Users and Value of Reduced
Productivity, by Age and Sex Cohort
(Number in thousands, $ in millions)

	Males		Females			Total	
				Employed	Housewives		
Age	Number	Value	Number	Value	Value	Number	Value
18-19	806	$ 1,313	601	$ 651	$ 140	1,407	$2,105
20-24	2,261	6,723	1,140	2,256	526	3,400	9,505
25-34	2,220	11,076	817	2,396	633	3,036	14,106
35-44	0	0	0	0	0	0	0
45-54	0	0	0	0	0	0	0
55-64	0	0	0	0	0	0	0
Total	5,286	$19,112	2,557	$5,303	$1,299	7,844	$25,716

Totals may not add due to rounding.

Source: Research Triangle Institute; special analysis of the 1982 National Household Survey (Miller et al., 1983).

Table III-24

Value of Lost Employment in 1980:
Drug Abusers in Residential Treatment
($ in millions)

Age	Males		Females		Total	
	Number	Value	Number	Value	Number	Value
18-19	1,206	$ 9	391	$ 3	1,597	$ 12
20-24	3,462	49	1,014	12	4,476	61
25-29	2,837	62	788	14	3,625	76
30+	3,015	79	588	11	3,603	89
Total	10,520	$199	2,781	$39	13,301	$238

Totals may not add due to rounding.

Source: Research Triangle Institute; verbal communication with NIDA.

Lost employment due to inpatient treatment cost an estimated $863 million in 1980, compared to an estimated $328 million in 1977. The prior study estimated that there were about 19,000 adults in residential facilities. The most recent data from CODAP indicate that nearly 39,000 adults received residential treatment in 1980. The remainder of the change can be attributed to wage inflation.

It is estimated that there were 6,000 adults with severe/profound mental retardation attributable to fetal alcohol syndrome. The expected productivity of these persons was $104.3 million (see the appendix, table B-4).

2. Drug Abuse

Reduced Productivity

Drug abuse is hypothesized to impair productivity of workers in a manner similar to the impact of alcohol abuse. State-of-the-art analysis reported in the appendix supports this contention.

It was found that individuals who reported that they had ever used marijuana daily for at least a month had household incomes 27.9 percent lower than those of persons with similar characteristics who had not used marijuana. The prevalence rates reported in table III-22, which indicate 7.8 million young men and women aged 18-34 had used marijuana at that level. The number in each age/sex group of table III-23 was multiplied by the (1) labor force participation rate (to adjust for persons unlikely to pursue employment), (2) age/sex productivity rates, and (3) impairment rate (27.9 percent) to obtain a value for reduced market productivity of $25.7 billion.

The prior study estimated that 17 percent of males aged 18-24 had an marijuana abuse-related productivity impairment of 14 percent (identical to the alcohol abuse impairment rate in that study), a cost of $2.8 billion. This study has found a prevalence rate of approximately 21 percent for males aged 18-24, and an impairment rate of 27.9 percent, twice that of the previous study. This study has included 12.5 percent of males aged 25-34 and approximately 8 percent of females aged 18-34 with productivity losses due to marijuana abuse. These latter estimates were not previously made.

Lost Employment

This figure has increased only marginally, from $223 million in 1977 to $238 million in 1980 (table III-24). The growth of 6.7 percent is much smaller than the increase in wages of 31 percent over the period. The difference is attributable to a decrease of over 20 percent in the population of drug abusers in residential facilities between 1977 and 1980.

3. Mental Illness

Estimates of indirect morbidity costs for mental illness increased with inflation and population growth over the 1977-80 period. Costs of partial disability grew from $1.6 billion in 1977 to $3.1 billion in 1980, while costs of complete disability grew from $12.7 to $16.8 billion. Losses due to institutionalization of the mentally ill were estimated at $1.3 billion in 1977. Updating these estimates for the effects of inflation yielded a 1980 estimate of $1.7 billion for this component (see tables III-25 to III-27).

Potentially, the largest component of economic cost from mental illness is that of reduced productivity in the work force. These costs are potentially of the same magnitude as estimates of reduced productivity due to alcohol abuse. Unfortunately, at the present time the lack of good data makes it difficult to assess productivity impairments associated with diagnoses of mental illness.

F. Other Related Direct Costs

1. Motor Vehicle Crashes

These costs increased from $1.8 billion in 1977 to $2.2 billion in 1980 (table III-28). This was an increase of 22 percent, which was several percentage points below the general inflation rate of 27.6 percent over the same time period.

The cost estimates are based on a recently completed study by the U.S. Department of Transportation (U.S. DOT, NHTSA, 1983) of the costs of motor vehicle crashes. These estimates used the causal factors developed by Berry and Boland and used in the prior RTI study.

2. Crime

Criminal justice system costs related to alcohol abuse increased from $1.5 billion in 1977 to an estimated $2.1 billion in 1980, an increase of 39.4 percent (table III-29). The largest components of this increase were felonious assault (which includes various types of interpersonal violence such as spouse and child abuse), driving under the influence, and public drunkenness.

Table III-25

Reduced Productivity in 1980: Mental Illness, Partial Work Disability, Number of Persons and Value of Reduced Productivity (Number in thousands, $ in millions)

Age	Males		Females			Total	
				Employed	Housewives		
	Number	Value	Number	Value	Value	Number	Value
18-19	16	31	10	$ 12	$ 3	26	$ 46
20-24	40	138	24	56	13	64	207
25-34	75	441	73	252	68	148	760
35-44	58	403	78	271	66	135	740
45-54	71	483	85	264	71	156	818
55-64	64	319	81	158	73	145	551
Total	324	$1,814	350	$1,013	$294	674	$3,122

Totals may not add due to rounding.

Source: Research Triangle Institute; Rehab Group Inc. (1979).

Table III-26

Lost Employment in 1980:
Mental Illness, Complete Work Disability,
Number of Persons and Value of Lost Employment
(Number in thousands, $ in millions)

			Females				
	Males			Employed	Housewives	Total	
Age	Number	Value	Number	Value	Value	Number	Value
18-19	11	$ 85	10	$ 51	$ 11	21	$ 147
20-24	27	377	24	230	54	51	661
25-34	66	1,611	78	1,090	293	143	2,994
35-44	62	1,750	84	1,197	290	145	3,238
45-54	113	3,133	168	2,129	576	281	5,838
55-64	102	2,066	160	1,283	594	262	3,942
Total	380	$9,022	523	$5,981	$1,818	903	$16,821

Totals may not add due to rounding.

Source: Research Triangle Institute; Rehab Group Inc. (1979).

Table III-27

Lost Employment in 1980: Mental Illness
Number of Adults (aged 16-64) Institutionalized
and Value of Lost Employment
(Number in thousands, $ in millions)

Type of Institution	Males		Females		Total	
	Number	Cost	Number	Cost	Number	Cost
Nursing Homes	76.5	$ 328	90.7	$286	167.2	$ 614
Psychiatric Hospitals	31.1	732	25.4	375	56.7	1,107
Total	107.8	$1,060	116.1	$661	223.9	$1,721

Totals may not add due to rounding.

Source: U.S. DOC, BOC (1979); unpublished data from NIMH (U.S. DHHS, NIMH, 1981); U.S. DOL, BLS, Monthly Labor Review, 1983.

Table III-28

Motor Vehicle Crash Costs Due to Alcohol Abuse, 1980
($ in millions)

Cost Category	Fatalities	Minimum/ Moderate Injury	Severe/ Critical Injury	Property Damage Only	Total
Legal/court costs	$ 684	$2,006	$ 804	$ 370	$3,865
Insurance administration	641	2,044	693	4,050	7,427
Accident investigation	7	149	21		176
Vehicle damage	174	3,268	557	16,984	20,983
Total	$1,505	$7,467	$2,075	$21,404	$32,451
Percent caused by alcohol abuse	37.0	10.5	9.75	3.0	
Total costs caused by alcohol abuse	$ 557	$ 784	$ 202	$ 642	$2,185

Totals may not add due to rounding.

Source: Research Triangle Institute; U.S. DOT, NHTSA, 1983; Berry and Boland, 1977.

Table III-29

Alcohol Abuse: Other Related Direct Costs - Public Financed Criminal Justice System, 1980
($ in millions)

Offense Types	(1) Causal Factor[1]	(2) Percent of All Persons Arrested for Nonalcohol Offenses[2]	(3) Police Protection Costs (PPC) (1x2xtotal PPC)[3]	(4) Percent of All Persons Charged for Nonalcohol Offenses[4]	(5) Legal and Adjudication Costs (LAC) (1x4xtotal LAC)[5]
Homicide	0.300	0.3	$ 15	0.2	$ 4
Felonious assault	0.269	4.2	184	3.5	65
Robbery	0.039	2.0	13	1.7	5
Burglary	0.047	6.9	53	7.1	23
Larceny	0.038	16.1	100	18.9	49
Auto theft	0.046	1.9	14	2.1	7
Driving under the influence	1.000	1,426,700[6]	31	1,141,360	25
Liquor law	1.000	463,500[6]	10	370,800	8
Public drunkenness	1.000	1,125,800[6]	25	900,640	20
Total			$445		$206

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[1] See text for a discussion of causal factors.

[2] Federal Bureau of Investigation (U.S. DOJ, FBI, 1981). See text for discussion of alcohol-defined offense categories.

[3] Bureau of Justice Statistics (U.S. DOJ, BJS, 1983). PPC = police protection expenditures plus proportionate allotments of other criminal justice costs. Total PPC = $16,274 million.

[4] Federal Bureau of Investigation (U.S. DOJ, FBI, 1978). Number of persons charged in alcohol-defined offense categories is 80 percent of those arrested for these offenses; see text.

[5] Bureau of Justice Statistics (U.S. DOJ, BJS, 1983). LAC = Judicial plus legal services plus public defense plus a proportionate allotment of other criminal justice costs. Total LAC = $6,861 million.

[6] See text for explanation of why arrest and charging frequencies are indicated instead of percentages of persons arrested and persons charged. The PPC and LAC costs for alcohol offenses were each estimated to be $21.84.

Table III-29 (continued)

Offense Types	(6) Percent of State and Federal Inmates[7]	(7) State and Federal Correctional Costs (SCC) (1x6xtotal SCC)[8]	(8) Percent of All Persons Charged[9]	(9) Local Correctional Costs (LCC) (1x8xtotal LCC)[10]	(10) Total Public Criminal Justice Costs (Alcohol) (3+5+7+9)
Homicide	17.6	$255	0.10	$ 1	$ 275
Felonious assault	12.6	164	2.30	16	429
Robbery	24.9	47	1.20	1	66
Burglary	18.1	41	4.70	6	123
Larceny	4.8	9	1.30	1	159
Auto theft	1.9	4	1.40	2	26
Driving under the influence	0.0	0	10.70	274	330
Liquor law	0.0	0	5.90	151	169
Public drunkenness	0.0	0	17.20	440	485
Total		$520		$892	$2,062

[7] Bureau of Justice Statistics (U.S. DOJ, BJS, 1982). Few individuals are incarcerated in state and Federal institutions for alcohol-defined offenses.

[8] Bureau of Justice Statistics (U.S. DOJ, BJS, 1983). Other criminal justice costs have been proportionately distributed. Total SCC = $4,847 million.

[9] Federal Bureau of Investigation (U.S. DOJ, FBI, 1978).

[10] Bureau of Justice Statistics (U.S. DOJ, BJS, 1983). Other criminal justice costs have been proportionately distributed. Total LCC = $2,558 million.

Totals may not add due to rounding.

Source: Research Triangle Institute.

There is no information to indicate that the causal relationship of alcohol abuse to criminality has changed since the prior report. Therefore, the assumptions for the causal factors used in the previous study have been used for this effort.

The estimate of direct criminal justice system costs in 1980 incorporates a change in methodology. Recent studies have implicated drug traffic in homicide and other violent crimes (this is discussed in the appendix). The new estimates reflect a 10 percent causal factor for drug abuse in homicide and assault, responsible for an increase of $250 million in the criminal justice system costs. Total direct criminal justice system costs due to drug abuse increased by 33 percent over three years, from $3.3 billion to $4.5 billion (table III-30). The low rate of increase was primarily due to a reduction in expenditures related to the enforcement of the drug laws. Drug related arrests declined as a proportion of all arrests during this period. Drug law offenders made up a smaller proportion of all prison inmates in the most recent prison survey (1978) than in the prior survey (1974).

Another change is introduced for mental illness (see table III-31). Persons arrested for the minor offenses of disorderly conduct, vagrancy, and others are often found to be mentally incompetent or ill and become the responsibility of the criminal justice system. This is also discussed in the appendix.

Private expenditures for legal services and protection against crime, which are estimated as a proportion of public expenditures, are relatively small. It has been estimated that private protection expenditures are 56.4 percent of public expenditures on police protection (Goldman, 1978). This suggests that alcohol abuse related private protection expenditures were $251 million in 1980 compared to $177 million in 1977 (table III-32). Correspondingly, drug abuse related private protection expenditures grew from $1.0 billion to $1.3 billion in 1980. The mental illness cost was $227 million.

Private expenditures on legal defense were also relatively small. For alcohol abuse, this cost component was estimated at $10 million in 1980. The comparable figures were $48 million for drug abuse and $8 million for mental illness.

Table III-30

Drug Abuse: Other Related Direct Costs - Public Financed Criminal Justice System, 1980
($ in millions)

Offense Types	(1) Causal Factor[1]	(2) Percent of All Persons Arrested for Nonalcohol Offenses[2]	(3) Police Protection Costs (PPC) (1x2xtotal PPC)[3]	(4) Percent of All Persons Charged for Nonalcohol Offenses[4]	(5) Legal and Adjudication Costs (LAC) (1x4xtotal LAC)[5]
Homicide	0.010	0.3	$ 5	0.2	$ 1
Felonious assault	0.010	4.2	68	3.5	24
Robbery	0.268	2.0	87	1.7	31
Burglary	0.224	6.9	252	7.1	109
Larceny	0.186	16.1	487	18.9	241
Auto theft	0.186	1.9	56	2.1	27
Stolen goods	0.186	1.7	51	1.3	17
Prostitution	0.128	1.2	25	1.0	9
Drug law	1.000	7.8	1,269	7.5	515
Total			$2,300		$974

[1] See text for a discussion of causal factors.

[2] Federal Bureau of Investigation (U.S. DOJ, FBI, 1981).

[3] Bureau of Justice Statistics (U.S. DOJ, BJS, 1983). PPC = police protection expenditures plus proportionate allotments of other criminal justice costs. Total PPC = $16,274 million.

[4] Federal Bureau of Investigation (U.S. DOJ, FBI, 1978).

[5] Bureau of Justice Statistics (U.S. DOJ, BJS, 1983. LAC = Judicial plus legal services plus public defense plus a proportionate allotment of other criminal justice costs. Total LAC = $6,861 million.

Table III-30 (continued)

Offense Types	(6) Percent of State and Federal Inmates[6]	(7) State and Federal Correctional Costs (SCC) (1x6xtotal SCC)[7]	(8) Percent of All Persons Charged[8]	(9) Local Correctional Costs (LCC) (1x8xtotal LCC)[9]	(10) Total Public Criminal Justice Costs (Drugs) (3+5+7+9)
Homicide	17.6	$ 84	0.1	$ 1	$ 91
Felonious assault	12.6	61	2.3	6	159
Robbery	24.9	323	1.2	8	449
Burglary	18.1	197	4.7	27	585
Larceny	4.8	43	1.3	6	777
Auto theft	1.9	17	1.4	7	107
Stolen goods	.01	0	0.9	4	72
Prostitution	.01	0	0.7	2	36
Drug law	5.7	276	5.0	128	2,178
Total		$1,001		$189	$4,454

[6] Bureau of Justice Statistics (U.S. DOJ, BJS, 1982).

[7] Bureau of Justice Statistics (U.S. DOJ, BJS, 1983). Other criminal justice costs have been proportionately distributed. Total SCC = $4,847 million.

[8] Federal Bureau of Investigation (U.S. DOJ, FBI, 1978).

[9] Bureau of Justice Statistics (U.S. DOJ, BJS, 1983). Other criminal justice costs have been proportionately distributed. Total LCC = $2,558 million.

Totals may not add due to rounding.

Source: Research Triangle Institute.

Table III-31

Mental Illness: Other Related Direct Costs - Public Financed Criminal Justice System, 1980
($ in millions)

Offense Types	(1) Causal Factor[1]	(2) Percent of All Persons Arrested for Nonalcohol Offenses[2]	(3) Police Protection Costs (PPC) (1x2xtotal PPC)[3]	(4) Legal and Adjudication Costs (LAC) (1x2xtotal LAC)[4]	(5) Local Correctional Costs (LCC) (1x2xtotal LCC)[5]	(6) Total Public Criminal Justice Costs (Drugs) (3+5+7+9)
Disorderly conduct	.10	7.4	$120	$ 51	$19	$190
Vagrancy	.10	0.3	5	2	1	8
Other offenses	.10	17.0	277	117	43	437
Total			$402	$170	$ 63	$635

[1] See text for a discussion of causal factors.

[2] Federal Bureau of Investigation (U.S. DOJ, FBI, 1981).

[3] Bureau of Justice Statistics (U.S. DOJ, BJS, 1983). PPC = police protection expenditures plus proportionate allotments of other criminal justice costs. Total PPC = $16,274 million.

[4] Bureau of Justice Statistics (U.S. DOJ, BJS, 1983). LAC = Judicial plus legal services plus public defense plus a proportionate allotment of other criminal justice costs. Total LAC = $6,861 million.

[5] Bureau of Justice Statistics (U.S. DOJ, BJS, 1983). Other criminal justice costs have been proportionately distributed. Total LCC = $2,558 million.

Totals may not add due to rounding.

Source: Research Triangle Institute.

Table III-32

Private Expenditures Related to Crime in 1980
($ in millions)

	Protection		Legal Defense		Total Private
	Public	Private[a]	Public	Private[b]	
Alcohol Abuse	$ 445	$ 251	$ 20	$10	$ 261
Drug Abuse	2,300	1,297	95	48	1,345
Mental Illness	402	227	16	8	235
Total	$3,147	$1,775	$131	$66	$1,841

[a] Calculated as 56.4 percent of public expenditures.

[b] Calculated as 50.7 percent of public expenditures.

Source: U.S. DOJ, BJS (1983), Goldman (1978), analysis by Research Triangle Institute.

Another element of crime costs is due to destruction of property. This value has grown from $15 to $24 million for alcohol abuse, and from $73 to $111 million for drug abuse (table III-33). All data are from the National Crime Survey program (U.S. DOJ, BJS, 1984). The causal factors have remained the same.

3. Other Direct Costs

The estimated costs of social welfare programs in 1980 were $201 million for mental illness, $38 million for alcohol abuse and $2 million for drug abuse (table III-34). The prior estimates (for 1977) were reported as $548 million, $142 million, and $12 million respectively. This represents an apparent decrease of substantial proportion over the 3-year period.

Using the same data sources as for the 1980 estimates, the figures for 1977 were reassessed at $184 million, $35 million, and $2 million, respectively. The growth over 3 years between 1977 and 1980, then, was 9.2 for mental illness, nothing for alcohol abuse, and 8.6 percent for drug abuse. The increased cost in administration of all funds listed in table III-34 was 16 percent($3.2 billion to $3.7 billion). For purposes of comparison, the revised figures for 1977 are included in table III-35.

From 1977 to 1980, fire losses, fire protection and highway safety expenditures attributed to alcohol abuse increased from an estimated $832.0 million to $1.23 billion, an increase of 48 percent(table III-36). Expenditure data for 1980 were obtained from the National Fire Protection Association(Karter, 1981) and the Statistical Abstract of the United States (U.S. DOC, BOC, 1982).

The fetal alcohol syndrome caused extra expenditures of $1.7 billion for special education, training, and rehabilitation programs. These estimates are highly contingent on the estimated prevalence of FAS in juvenile and adult cohorts, which must be confirmed in future studies. See the appendix for a discussion of how these costs were estimated. This value was not estimated previously.

There was a 73 percent increase in drug traffic control expenditures between 1977 and 1980, from $311 million to $537 million. Expenditures for 1980 were obtained from a report prepared for the President by the Strategy Council on Drug Abuse (Drug Abuse Policy Office, 1982).

Table III-33

Property Destruction Due to Crime in 1980
($ in millions)

Crime	Value	Causal Factor		Value	
		Alcohol Abuse (percent)	Drug Abuse (percent)	Alcohol Abuse	Drug Abuse
Robbery	$ 12	3.9	26.8	$ 0.5	$ 3.2
Rape	3	26.9	0.0	0.8	-
Personal Larceny	206	3.8	18.6	7.8	38.3
Burglary	151	4.7	22.4	7.1	33.8
Household Larceny	87	3.8	18.6	3.3	16.2
Motor Vehicle Theft	106	4.6	18.6	4.9	19.7
Total	$565	-	-	$24.4	$111.2

Source: Unpublished data from the U.S. DOJ, BJS, 1984.

Table III-34
Public Program Social Welfare Administrative Expenditures and Costs Due to ADM Problems, 1980
($ in millions)

Program	Total Expenditures①	Percent Administrative Costs	Total Administrative Costs①	Costs Alcohol Abuse Percent	Costs Alcohol Abuse Amount	Costs Drug Abuse Percent	Costs Drug Abuse Amount	Costs Mental Illness Percent	Costs Mental Illness Amount
OASDHI disability payments	$15,437	2.4	$ 370	3.4	$12	③	$ ②	8.9	$ 33
Unemployment insurance	16,503	11.0	1,815	-	-	-	-	-	-
Railroad temporary disability insurance	64	7.8	5	3.4	②	③	②	8.9	2
State temporary disability insurance④	1,330	3.2⑥	43	3.4	1	③	②	8.9	4
Workers compensation④	9,588	4.4⑥	422	-	-	-	-	-	-
Public assistance⑤	1,219	15.3	186	4.6	9	0.5	1	4.7	9
Supplemental security income	7,446	8.8	655	0.8	5	③	②	18.8	123
Food stamps	644	4.4	28	4.8	1	0.5	②	4.7	1
Veteran's pensions and compensation	11,306	0.8	90	9.7	9	0.9	1	23.6	21
Vocational rehabilitation④	976	4.7⑥	46	-	-	-	-	19.3	9
Total	$64,513		$3,661		$38		$2		$201

①Source: Social Security Administration, Office of Retirement and Survivors Insurance, personal communication.

②Less than $.5 million.

③Less than .0005.

④Excludes hospital and medical benefits.

⑤Excludes vendor medical payments and social services.

⑥Percent does not exclude hospital and medical program administrative costs.

(-) No causal relationship is assumed, or no beneficiaries reported ADM as primary cause of eligibility.

Totals may not add due to rounding.

Source: Research Triangle Institute.

Table III-35
Public Program Social Welfare Administrative Expenditures and Costs Due to ADM Problems, 1977
($ in millions)

Program	Total Expenditures①	Percent Administrative Costs	Total Administrative Costs①	Costs Alcohol Abuse Percent	Costs Alcohol Abuse Amount	Costs Drug Abuse Percent	Costs Drug Abuse Amount	Costs Mental Illness Percent	Costs Mental Illness Amount
OASDHI disability payments	$11,463	3.5	$ 401	3.4	$ 14	③	②	8.9	$ 36
Unemployment insurance	13,823	11.8	1,631	-	-	-	-	-	-
Railroad temporary disability insurance	79	4.9	4	3.4	②	③	②	8.9	②
State temporary disability insurance④	966	3.3	32	3.4	1	③	②	8.9	3
Workers compensation④	5,902	4.0	236	-	-	-	-	-	-
Public assistance⑤	1,112	13.7	152	4.4	7	0.7	1	3.0	5
Supplemental security income	6,240	8.5	530	0.8	4	③	②	18.8	100
Food stamps	376	7.8	29	4.4	1	0.7	②	3.0	1
Veteran's pensions and compensation	9,002	0.8	73	11.8	9	1.1	1	36.2	26
Vocational rehabilitation⑥	1,007	6.5	65	-	-	-	-	20.9	14
Total	$50,050		$3,154		$35		$2		$184

①Source: Social Security Administration, Office of Retirement and Survivors Insurance, personal communication.

②Less than $.5 million.

③Less than .0005.

④Excludes hospital and medical benefits.

⑤Excludes vendor medical payments and social services.

⑥Percent does not exclude hospital and medical program administrative costs.

(-) No causal relationship assumed.

Totals may not add due to rounding.

Source: Research Triangle Institute.

Table III-36

Additional Other Related Direct Costs Due to Alcohol and Drug Abuse and Mental Illness, 1980 ($ in millions)

Cost Element	Total Expenditures	Source	% Due to Disorder	Amount
Alcohol Abuse				$1,229
Fire losses	$6,254	Karter (1981)	6.1	381
Fire protection	5,718	Statistical Abstract of U.S. (U.S. DOC, BOC 1982)	11.2	640
Highway safety	1,126	Statistical Abstract of U.S. (U.S. DOC, BOC 1982)	18.5	208
Drug Abuse				$537
Drug traffic control	537	Drug Abuse Policy Office (1982)	100.0	537
Mental Illness				$659
Education	659	Frank and Kamlet (1984)	100.0	659

Source: Research Triangle Institute and references in above table.

In addition, a value of $659 million has been included for special care of the mentally ill in the education system. This value was produced by Frank and Kamlet (1984). No figure was estimated in 1977.

G. Other Related Indirect Costs

1. Lost Work Time Due to Crime

Victims of crime often are uncompensated, even though they experience short term hardships from their victimization. This represents an important cost to the victim which has no offsetting transfer. The National Crime Survey performs victimization surveys to evaluate the extent of this hardship. They estimated the total number of victimizations for 1980 and work time lost (U.S. DOJ, BJS, 1984).

Using the crime/drug and crime/alcohol attribution factors developed in the 1981 report, the value of victim time by type of crime has been calculated (see table III-37). The value for drug abuse property crime was $845 million, and for the violent crime of felonious assault it was $74 million. These same figures for alcohol abuse were $172 million and $198 million.

2. Crime Careers: Drug Abuse

These costs increased from $5.1 billion in 1977 to $8.7 billion in 1980 (table III-38). There are several reasons for this large increase. First, the number of opiate addicts was estimated to have increased by about 10 percent, from 450,000 in 1977 to 492,000 in 1980. Secondly, wage inflation was greater than 30 percent over the three-year span.

For heroin addicts, these costs reflect the time spent engaged in crime, rather than legal employment, motivated primarily by their addiction. Studies of this population generally find that heroin addicts commit a large number of income-generating crimes, but more and more surveys also find that these individuals often have jobs. The Treatment Outcome Prospectives Survey (TOPS) (Rachal, Hubbard, Cavanaugh, Bray, Collins, Allison, and Craddock, 1981) has found that while large proportions of opiate abusers report illegal activities as their major source of income, it is not their only income source. In fact, a significant proportion report jobs as a primary activity.

For estimation of the lost productivity due to crime careers of heroin addicts, we distributed the estimated population of 492,000 in 1980 across sex and age groups in proportion to CODAP (U.S. DHHS, NIDA, 1981) admissions of opiate abusers. Analysis of TOPS data indicates that in the 1979 cohort

Table III-37

Lost Work Time Due to Crime in 1980: Number of Persons and Value of Reduced Productivity

Type	Number[a] (thousands)	Average Work Days Lost[b]	Proportion of Offenses Due to: Alcohol Abuse (percent)	Proportion of Offenses Due to: Drug Abuse (percent)	Value Attributed to: Alcohol Abuse ($ in millions)	Value Attributed to: Drug Abuse ($ in millions)
Rape	171	3.3	26.9	10.0	$(11)[c]	$ (4)[c]
Assault	4,732	2.1	26.9	10.0	(187)[c]	(70)[c]
Robbery	1,038	3.1	3.9	26.8	9	61
Burglary	6,704	2.0	4.7	22.4	45	215
Larceny	26,402	1.5	3.8	18.6	108	527
Auto Theft	1,365	2.3	4.6	18.6	10	42
Total	40,412	14.3	-	-	$172	$845

[a] Data from 1978 victimization series.

[b] Value of average daily productivity (employment plus household) in 1980 was $21,675 for males, $14,798 for females, or an average of $70 per day for both.

[c] These values have been included as lost employment due to violent crimes under core indirect costs in table III-19.

Source: Unpublished data, U.S. DOJ, BJS, 1984.

Table III-38

Drug Abuse Costs of Lost Productivity Due to Crime Careers in 1980:
Number of Persons Foregoing Legitimate Productive Pursuits and Value of Loss
($ in millions)

Age	Opiates						Nonopiates						Total	
	Males		Females		Total		Males		Females		Total			
	Number	Value	Number	Value	Number	Value	Number	Value	Number	Value	Number	Value	Number	Value
< 18	1,207	$ 0	884	$ 0	2,091	$ 0	88,509	$ 0	49,394	$ 0	137,902	$ 0	139,993	$ 0
18-19	3,839	16	3,170	8	7,009	24	54,397	195	19,574	36	73,972	231	80,981	255
20-24	48,889	375	31,422	143	80,312	518	121,099	792	38,608	131	159,706	922	240,017	1,440
25-29	128,195	1,506	53,362	365	181,557	1,872	75,634	757	25,946	132	101,580	889	283,136	2,761
30-44	158,925	2,381	41,580	288	200,505	2,669	69,301	884	24,560	127	93,861	1,011	294,365	3,680
> 44	17,383	271	3,144	20	20,527	291	19,605	260	7,630	37	27,235	297	47,762	589
Total	358,432	$4,550	133,562	$825	492,000	$5,374	428,545	$2,888	165,711	$462	594,256	$3,350	1,086,256	$8,725

Source: Research Triangle Institute; U.S. DHHS, NIDA, CODAP (1981).

54 percent of male opiate abusers reported crime as their major source of income, as did 39 percent of the females. These proportions were assumed to be involved in crime full time, consequently putting no time into productive activity. The value of this time was estimated at $5.374 billion in 1980.

The other large contribution to the increase in this cost component was an increase in the estimated size of the nonopiate drug abuser population. To make a lower bound estimate of the entire nonopiate user population, it was assumed that nonopiate drug users are as likely to require treatment during the course of a year as opiate addicts. Of the estimated 1980 opiate addict population of 492,000, 115,000 were admitted to treatment for a penetration rate of 23 percent. Assuming that the 23 percent penetration rate applies to the nonopiate user population, the 139,000 admissions of nonopiate users reported in CODAP in 1980 would come from a population of nearly 600,000 nonopiate users. The large increase in productivity losses due to crime careers is attributable to the 65 percent increase since 1977 in nonopiate users' admissions to drug abuse treatment.

Some perspective is gained on these estimates by recalling that the ECA study (Myers et al., 1983) yielded a rough estimate that 1 percent of the population surveyed were drug dependent. Application of this rate to the 162.8 million adults found in the 1980 Census predicts a total of 1.628 million drug dependent persons. NIDA estimates that there were 492,000 heroin addicts in 1980, leaving about 1.136 million nonopiate drug dependent persons in 1980. This estimate is likely to be too high because the early ECA survey results are from urban areas. The NIDA national surveys have consistently found lower prevalence of drug abuse in smaller urban and rural areas than in large urban areas. Given these caveats, the estimates of 600 thousand nonopiate drug abusers may be considered conservative.

Nonopiate users also engage in income-generating crime, although not to the extent that heroin addicts do. TOPS has reported that 46 percent of the male and 29 percent of the female nonopiate abusers claimed that crime was their major source of income. Again, these proportions of the population are assumed to be entirely involved in crime and not engaged in market or household activity. When applying sex- and age-specific productivity rates, the lost productivity was estimated at $3.35 billion for 1980.

3. Incarceration

The productivity of incarcerated individuals is usually entirely lost to society. The value of this cost component for alcohol abuse increased to $1.8 billion in 1980, a 27 percent increase over the 1977 value of $1.4 billion (table III-39). Lost productivity from incarceration due to drug abuse increased from $1.3 million in 1977 to an estimated $1.5 million in 1980. The prior study estimated that nearly 38,000 persons were incarcerated during 1977 for violation of drug laws, compared with a 1980 estimate of 26,000, a decrease of almost 12,000. The 1977 estimates were developed from data from the 1974 Census of Prisons and Jails, while the 1980 estimates of incarceration were developed from data in the 1978 Census of Prisons and Jails, published in 1982.

4. Motor Vehicle Crashes: Alcohol Abuse

The 1977 estimate of $354 million was inflated to $464 million based on the inflation factor for wages and salaries. There are no data yet with which to estimate the number and cost of drug-related motor vehicle crashes.

Table III-39

Lost Productivity Due to Incarcerations in 1980: Number and Proportions of Incarcerations Due to Alcohol and Drug Abuse and Value of Loss ($ in millions)

Offense	Adults Incarcerated Due to Offense in: Federal & State Prisons	Local Jails	Total	Proportion of Offenses Due to: Alcohol Abuse	Drug Abuse (percent)	Mental Illness	Person Years Served in 1980 for Crime Due to: Alcohol Abuse	Drug Abuse (person years)	Mental Illness	Value Attributed to: Alcohol Abuse	Drug Abuse ($ in millions)	Mental Illness
Homicide	53,059	183	53,242	30.0	10.0	0.0	15,625	5,200	0	$ 311	$ 104	$ 0
Felonious assault	37,985	4,212	42,197	26.9	10.0	0.0	11,099	4,126	0	220	82	0
Robbery	75,066	2,197	77,264	3.9	26.8	0.0	2,947	20,254	0	59	403	0
Burglary	54,566	8,607	63,173	4.7	22.4	0.0	2,903	13,835	0	57	274	0
Larceny	14,471	2,381	16,851	3.8	18.6	.0.0	626	3,064	0	12	61	0
Auto theft	5,728	2,564	8,292	4.6	18.6	0.0	373	1,507	0	7	30	0
Drug laws	17,184	9,156	26,340	0.0	100.0	0.0	0	25,729	0	0	503	0
Receiving stolen property	301	1,640	1,950	0.0	18.6	0.0	0	353	0	0	7	0
Prostitution	301	1,202	1,503	0.0	12.8	0.0	0	198	0	0	4	0
Driving under the influence[a]	0	19,594	19,594	100.0	0.0	0.0	19,006	0	0	359	0	0
Liquor laws	0	10,804	10,804	100.0	0.0	0.0	10,524	0	0	198	0	0
Public drunkenness	0	31,497	31,497	100.0	0.0	0.0	30,680	0	0	577	0	0
Disorderly Conduct	0	13,500	13,500	0.0	0.0	10.0	0	0	1,350	0	0	26
Vagrancy	0	539	539	0.0	0.0	10.0	0	0	54	0	0	1
All Other Offenses	0	31,140	31,140	0.0	0.0	10.0	0	0	3,114	0	0	61
Other	42,809	43,819	86,628	0.0	0.0	0.0	0	0	0	0	0	0
Total	301,470	103,124	404,594	-	-	-	93,863	74,273	4,518	$1,801	$1,466	$88

[a] Due to the exceedingly low proportion of prison sentences for persons arrested for driving while under the influence of alcohol, the Census of Prisons did not report this as an individual category.

Totals may not add due to rounding.

Source: Research Triangle Institute; U.S. DOJ., BJS, Sourcebook of Criminal Justice Statistics (1983).

IV. FRAMEWORK FOR EXTENSION OF RESULTS TO PROGRAM EVALUATION AND PUBLIC POLICY DECISIONMAKING

The results of this study indicate that ADM has major impacts on the economic well-being of our society, due to the costs associated with both the undesirable consequences of ADM and society's efforts to address these disorders. The total value of all costs in 1980 was $190.7 billion, or $89.5 billion for alcohol abuse, $46.9 billion for drug abuse, and $54.2 billion for mental illness. In the health sector alone, expenditures on personal health services were $31.6 billion, with another $3.8 billion spent on research, training, and construction of facilities. These real expenditures of $35.4 billion were 14.3 percent of all health expenditures, and 1.3 percent of U.S. gross national product in 1980.

Cost benefit and cost effectiveness analyses (CBA and CEA) are naturally related to cost of illness (COI) studies such as the present one. COI estimates the total cost (economic impact) of a disorder by first identifying the tangible consequences of an illness, then quantifying their level of occurrence and, finally, assigning appropriate values to them. CBA uses the same three steps to indicate the relative effectiveness of intervention strategies at reducing total costs to society. The first step is to identify the consequences of the disorder which the intervention might effect. The second step is to quantify the effects, and the third step is to assign values to the different intervention effects so that expenses of the intervention (costs) can be compared to the value of the avoided consequences (benefits). The benefits and costs may be directly compared because they are expressed in common units. Cost benefit analysis (CBA) may be used to answer questions such as:

- How do the economic benefits to society compare to the costs of a new treatment regimen?
- Which of two or more alternative public strategies has a higher ratio of benefits to costs?
- What is the benefit or cost to society of increasing (or decreasing) the size of particular programs?
- Given some sum of money, which of several alternative programs will yield the greatest economic benefit to society?

In contrast, cost effectiveness analysis (CEA) has a narrower use. Its purpose is to find the cost of achieving a single dimension outcome. The outcome is expressed in physical terms such as improvement in health status, or number of patients treated, rather than being translated into dollars saved on treatment or economic loss associated with illness. The emphasis is on how to identify the least-cost program, intervention, or technique for achieving the specific objective. CEA answers questions such as:

- To save 5,000 alcohol-related deaths from motor vehicle crashes, is it least expensive to institute safety features in cars, reduce the speed limit, or increase law enforcement efforts?
- Can manic depressives be treated less expensively (with equal effectiveness) as inpatients or outpatients?

Of course, some impacts of public policies have no market value and can not be assigned a dollar value for incorporation into cost benefit studies. These intangible impacts of public policies may be more, less, or equally as important as economic benefits in shaping policy decisions. Whenever an economic cost benefit study is performed, it is essential that the noneconomic impacts of policies also be assessed so that policy decision-makers may weigh them simultaneously with economic benefits.

Increasing emphasis has been placed on cost benefit and cost effectiveness analysis in the past 15 years. Particular emphasis has been placed on use of these studies in health policymaking. National health expenditures grew from $42 billion in 1965 to $247 billion in 1980 (Gibson and Waldo, 1981). As a share of gross national product, health expenditures increased from 6 percent in 1965 to approximately 9.5 percent in 1980.

Interest in cost effectiveness and cost benefit analysis has been further spurred by the rapid advances made in medical technology. Organ transplants have been performed successfully and failing organs have been successfully assisted by technologies such as kidney dialysis, respirators, and now potentially artificial hearts. Increasingly complex technology is often very expensive.

Probably a greater motivation for the federal government's interest in cost benefit analysis is that its share of the nation's health bill has expanded dramatically, from 10.1 percent in 1965 to 28.7 percent in 1980 due to the expansion of the Medicare and Medicaid programs. The shares paid by state and local government (about 11 percent) and private health insurance (about 25 percent) remained roughly constant over the entire 15 year period though total dollars have increased. A recent study performed under the auspices of the Office of Technology Assessment (OTA) resulted in a multiple volume report that examined the theory and practice of cost benefit and cost effectiveness analysis as applied to a number of specific health problems (U.S. Congress, OTA, 1980a). The work produced general outlines for performance of CBA and CEA studies, as well as reviewing and producing some state-of-the-art studies for Congress.

A limited set of conclusions was offered in another study commissioned by OTA (Saxe, 1983) which examined the approaches to alcohol treatment, research on treatment benefits, costs and benefits of treatment, and reimbursement issues. The study concluded that results of evaluations performed to date are mixed.

The 1980 OTA study (U.S. Congress, OTA, 1980b) similarly concluded that though studies performed to date are not completely comparable due to differences in evaluation design, the costs and benefits examined, and the method of placing a value on those costs and benefits, it appears that psychotherapy has positive effects. The studies of drug abuse treatment reviewed in this volume were found to be in accord with psychotherapy CBA and CEA studies which have found favorable cost benefit ratios for treatment.

A useful product of the OTA studies was a set of ten general principles of analysis for CEA/CBA methodology. The principles are:

- Define problem
- State objectives
- Identify alternatives
- Analyze benefits/effects
- Analyze costs
- Differentiate perspective of analysis
- Perform discounting
- Analyze uncertainties
- Address ethical issues and
- Interpret results.

These principles establish a solid foundation upon which cost effectiveness and cost benefit analysis of intervention strategies for ADM problems can be based. An in-depth presentation of these principles can be obtained from the OTA study. This chapter will discuss how the framework and cost concepts used in this ADM cost-of-illness study fit into, and complement, the performance of cost benefit and cost effectiveness studies of ADM interventions. The remaining materials are organized around the 10 principles of the OTA study.

A. Define the Problem

A CBA or CEA should only be undertaken when there are alternative approaches to a problem that can be clearly and logically defined. Both CBA and CEA yield measures that compare the relative costs, benefits, and impacts of alternative strategies to addressing the problem. Where there are alternative strategies to address a well-defined problem, CBA and CEA allow them to be compared on economic criteria. Where there is only one way to address a particular problem, the problem is probably defined too narrowly and should be respecified.

The first step in performing a cost benefit or cost effectiveness analysis is to determine the scope of the problem and to define it at a level that is suitable to the intended intervention, program, or strategy which is to be analyzed. For instance, in a comprehensive study examining drug abuse policy alternatives, the problem might be "the economic burden of drug abuse on society." With such a broad definition of the problem, the analyst must examine a full range of consequences of drug abuse, starting with health problems and drug overdoses, proceeding to criminal careers, impacts on the criminal justice system, and incarceration of criminals, as well as the effect of drug abuse on productivity in the workforce. A more narrowly defined problem would be that of heroin addiction, with its attendant consequences. A still narrower problem definition would be the issue of marijuana use and its effect in the workplace.

A problem definition for alcohol abuse may have similar breadth or specificity. One might examine all of the cost-related consequences of alcohol abuse or only specific problems such as drinking and driving, brain dysfunction, chronic hepatitis, pancreatitis, or liver cirrhosis.

B. State Objectives

The objectives of the evaluation of a particular public policy should be clearly defined. The objectives may be very broad in scope, or very specific. A very broad scope objective may be "reduce the economic cost to society." More specific objectives can be used in evaluating similar intervention strategies. A narrowly defined objective would be to reduce the number of motor vehicle crash deaths due to alcohol abuse. Other objectives might be to increase enrollment of heroin addicts in treatment, or to encourage depressed persons to seek assistance.

A strategy would be evaluated vis-a-vis its impact on the consequences that can be assigned economic values such as utilization of health care treatment, premature mortality, productivity at home and in the workforce, and a series of other impacts including criminality and motor vehicle crashes.

C. Identify Alternatives

Many different approaches can be taken to reduce the impact of alcohol abuse, drug abuse, and mental illness. The CBA or CEA must specify which alternatives can be targeted to the defined scope of the problem and policy objectives. Evaluations should focus on those options that are most likely to directly achieve policy objectives. Table IV-1 lists various strategies employed to combat ADM in the United States. These range from inpatient and outpatient treatment to legal restrictions and sanctions, and to education and other prevention efforts.

There are a variety of strategies for reducing mental illness. These include treatment in assorted inpatient settings, including long-term and short-term facilities, and from a growing variety of outpatient care providers such as psychiatrists and psychologists social workers, counselors, and various therapists. Drug therapy has also been found useful in treating various mental illnesses, a notable example being the lithium treatment for manic depression.

A major technique for public intervention into mental illness occurs when the government pays some or all of the price for treatment from various providers. This occurs for individuals who are eligible for treatment under Medicare or Medicaid, for example. Subsidized services may also be obtained in some circumstances from community mental health centers and other free standing facilities. Of course, a significant proportion of the population is not eligible for subsidized health care services and must cover the expenses either through private insurance or out-of-pocket payments.

Table IV-1

Examples of ADM Public Strategies and Programs

- Subsidize special treatments with utilization at own option (may be subsidized by government or private entities)
  - Inpatient (long or short term)
    - State, county, or private mental hospitals
    - Intermediate term specialized facilities
    - Short term general facilities
  - Outpatient (various types of providers)
    - Psychiatrists
    - Psychologists
    - Social workers
    - Other (specialized or not)
  - Supplies (drugs or prostheses)
    - Methadone
    - Antabuse-disulfiram
    - Lithium
- Mandate specific treatment regimes
  - "Commitment" to inpatient facility
    - Health
    - Corrections
  - Probation to an outpatient program
    - Care of a professional (health or criminal justice)
    - Treatment at program
    - Complete education (drinking and driving)
- Supply reduction/restriction
  - Alcoholic beverage laws
    - Age restrictions
    - Zoning and licensing restrictions
    - License (and tax) production
  - Drug laws
    - Regulate/restrict domestic production
    - Drug interdiction
- Other deterrence
  - Differential penalties for alcohol-related consequences
  - Differential penalties for drug-related consequences
- Education and other prevention efforts
- Research and development

A range of strategies comparable to those for mental illness are offered for the addictive disorders. Strong emphasis has been placed on drug demand reduction strategies such as providing drug abuse treatment programs and preventing new "starts" through education. Substantial resources have been put into education and other prevention efforts intended to reduce the incidence and prevalence of substance abuse and to ultimately reduce their social consequences. For instance, there have been concerted efforts made through the various media to convey the message that drunk driving is a threat to self and society. A similar set of public service messages has been used to discourage drug abuse. Another example of prevention efforts is the information that is provided to young women about the association of birth defects and substance abuse.

Public policies that address alcohol and drug abuse also include supply reduction efforts for both alcohol and drug abuse. All levels of government, for example, are mandated to interdict the flow of illegal drugs in order is to reduce their availability and, presumably, to increase their price, discouraging consumption. Nonmedical consumption of a long list of psychoactive substances is also proscribed, with sanctions for possession, consumption, and distribution.

There is a comparable set of supply reduction laws for alcohol abuse. First of all, purchase, possession, and consumption of alcohol by those who are underage is forbidden by law. Rules regarding where, when and under what conditions alcohol can be purchased and consumed affect all drinkers. Secondly, alcoholic beverages are taxed, increasing the price and potentially reducing consumption. The manufacture of alcoholic beverages is licensed and regulated. Furthermore, retailers are subject to licensing and zoning laws.

D. Analyze Benefits and Effects

The economic benefits from any public intervention strategy are the reduction in unfavorable consequences of those disorders which can be assigned a market value. Avoiding the tangible consequences constitutes a benefit to society of the intervention strategy. A major contribution of the present study for cost benefit analysis is the enumeration and identification of a number of these consequences of the ADM disorders. These assigned economic values are shown in chapter III of this report. A short list of these is repeated in table IV-2.

Table IV-2

Benefits and Costs to be Assessed in CBA or CEA Analysis

- Quantifiable benefits and costs
  - Health consequences framework
    - Health treatment
    - Mortality
    - Morbidity
    - Property destruction (crime or accidents)
    - Criminal justice response
    - Victim's time
    - Crime career
    - Incarceration
    - Other
  - Other impacts
    - Transfers of income via social welfare programs
    - Amount/value of substances illicitly consumed
    - Crimes commited
      - Number by type
      - Value of property transferred
- Generally nonquantifiable benefits and costs
  - Pain and suffering
  - Bereavement
  - Psychosocial development
  - Familial health

Intervention strategies may reduce the need for health treatment services or for more expensive treatment strategies by persons with ADM problems. For example, the introduction of antidepressive drugs has allowed people to be treated as outpatients, thereby avoiding the cost of maintaining them in residential treatment facilities. Other benefits may include allowing persons to return to productive endeavors and to prevent suicide.

Another very significant impact of ADM is premature mortality. An intervention strategy that can reduce premature mortality can be said to avoid the loss in market and household productivity associated with those deaths. Some strategies may reduce the number of premature deaths due to alcohol-related trauma such as death from motor vehicle accidents, falls, fires, drownings, and other accidents. Such strategies would include public interventions (for example, identification of drinking drivers and referral to treatment, and changes in drinking age laws) as well as community and school based prevention efforts.

The ADM disorders can cause productivity losses through lost employment (while incapacitated either at an inpatient facility or at home) or reduced productivity while at work or in the home. Again, an intervention strategy that can improve the productivity of ADM victims or make it possible for them to rejoin the workforce "avoids" those costs. This avoidance is considered the benefit of the intervention.

Certainly, the backbone of any evaluation is establishing the effectiveness of an intervention. The impact of a program, strategy, or technology should be quantified along all consequences that it might affect. The nature, degree, and value of changes must be accurately measured and related to the size, duration, strength and cost of the intervention undertaken. The CBA or CEA must establish a clear causal link between the intervention and abatement of consequences that can be reasonably expected to be achieved at full scale operation. Evaluation designs need to be very rigorous to meet this requirement.

After identifying the potential benefits from intervention strategies is the equally important step of quantifying the benefits. The issues become: How much impact will an intervention strategy of a particular level have on the tangible consequences of the ADM problem? How much drug use will be avoided or mitigated by prevention efforts? Will intervention strategies result in fewer drug overdoses, fewer medical costs involving toxic doses, and fewer premature mortalities? Will the efforts eventually prevent people from abusing the most dangerous drugs at high frequencies, behaviors which are associated with impairments in productivity and psychological and physiological dependence?

Many of the benefits of prevention efforts effect future years as well as the present year. Alcohol abuse, drug addiction, and mental illness often are long-term problems with impacts over many years. Problem drinking may adversely affect a person's productivity over his/her entire lifetime. Prevention or treatment which is partially or completely successful may yield benefits over many years to the problem drinker and society. Preventing the start of a criminal career or drug use prevents future consequences as well as current consequences. Effective outpatient treatment for mental illness allows those in treatment to remain productively involved in society and yields long-term benefits that ought to be included in current evaluations of the alternative interventions. The expected future benefits from current interventions are vital for rigorous cost benefit studies.

Other benefits of ADM intervention strategies may be valued above and beyond their contribution to social costs. These include transfers of income through social welfare programs, the amount and value of psychoactive substances which are illicitly consumed, and the number of crimes committed due to ADM. It should be emphasized that each of these impacts has already been assigned a value, discussed above, that would constitute a benefit under the cost benefit criteria. However, additional aspects of these impacts are also important for policy evaluation and should be quantified for their additional contribution to the economic cost. Finally, even though the avoidance of intangible consequences such as the pain and suffering associated with ADM is not ordinarily assigned a value, these dimensions are affected to the extent that the incidence, prevalence, and consequences of ADM disorders are reduced.

E. Analyze Costs

So far, we have discussed the benefits of an intervention strategy. In determining the other part of the ratio, cost, the market cost of operating an intervention strategy should be used. These costs should include, not only the expense of providing direct services to individuals, but also the accruals, overhead expenses, and capital costs that are connected to the service.

Average and marginal costs must be distinguished in evaluating a program or a strategy. For existing programs, average cost data are most readily available. The average costs are equal to the total costs of operating a program at its current level divided by the units of services produced or delivered. This yields a cost per service unit. Marginal costs are most appropriate in evaluating the potential expansion or contraction of an existing program. Marginal cost is the anticipated cost for increasing or decreasing (but not eliminating) services produced or delivered.

Marginal cost analysis can be highly significant in studying the expansion or contraction of an inpatient or outpatient program. Such a program is likely to have large fixed costs for buildings, equipment, property, and administrative personnel. If the program has excess physical capacity and ample administrative support, an increase in the level of services delivered may not substantially affect any of these fixed factors but may only involve costs for additional treatment personnel and assorted supplies. Some treatment programs, however, may be at maximum capacity and expansion may only be possible by moving to bigger quarters, improving record systems, adding managerial staff, etc. A decreased level of services (that is, fewer clients and/or fewer units of service delivered), however, could raise the unit cost of services delivered because fixed costs often remain the same.

F. Differentiate Perspective of Analysis

The costs and benefits of an intervention strategy in ADM problems differ depending on whether they are viewed from the perspective of the person with the problem, the perspective of the people or organizations who contact that person, or the perspective of the society as a whole.

An important example of this principle occurs with employed substance abusers and employed mentally ill persons that do not obtain treatment for their ADM problem. Such persons are often unable to meet the demands of the work environment. Normally, when an employer first discovers an employee is a substance abuser or mentally ill, the problem is ignored. Sometimes workers leave "voluntarily" because they are unable to cope with the working environment, but if the problem persists and reprimands are ignored, the worker is fired. This is particularly true for lower-level jobs where the employer has invested little in training and where there is a ready pool of qualified replacements.

Unskilled jobs are disappearing. As unemployment rates improve, the pool of ready replacements is drying up. More and more, employers have a significant investment in developing the skills of an employee as well as indoctrinating the employee to firm-specific knowledge. Passively allowing an obviously troubled employee to resign or summarily firing a problem employee sacrifices this investment. Out of the expectation that the cost of rehabilitating an employee will be outweighed by the benefits to the company, employers are increasingly referring problem employees to treatment, allowing them to participate in treatment and, even, providing the treatment.

From society's vantage point, a program is considered to be justified if the benefits to society outweigh the social costs. For example, people who are terminated from their employment or who cannot find an employer willing to hire them because of an ADM problem constitute a social economic cost because their potential productivity is unused. Those who terminate often have firm-specific knowledge and experience that is lost when they terminate. Should they find new employment, they initially will not be as valuable to the new employer as to the old employer because their old knowledge is useless, and they do not yet have firm-specific knowledge for the new employer. The net contribution to society's productivity is, therefore, lowered.

A second reason that the corporate cost benefit calculation may differ from the social cost benefit calculation is that rehabilitation for substance abuse has been found to be more effective with employed individuals than unemployed individuals. Rehabilitation may be more difficult and, consequently, more costly to society for a person dismissed from work because of a substance abuse problem. Once again, the social cost benefit equation is different than that for the private corporation. These factors may be significant in evaluating whether society might support an intervention strategy for rehabilitation of workers with substance abuse problems: the social costs and benefits of such treatment regimens may be quite different from the private cost and benefits, which may justify public support for EAP services in private corporations.

In this study, social (rather than private or individual) economic costs are weighed. Voluntary and involuntary transfers of income are not part of that equation. Only the loss of resources while effecting the transfer and administration costs (or property damage) is a dead weight loss to all concerned. Nonetheless, it may be relevant to identify transfers that occur because policymakers may consider them significant factors in evaluating the impact of public policies. This is particularly the case when one particular segment of society (income group, racial group, or resident of a particular area such as cities) gains or loses at the expense of another segment.

The net value of income transferred to recipients does not constitute a cost from society's perspective. The income received by beneficiaries for their use has been simply transferred to them from the taxpayers. These transfers occur through public subsidization of treatment services, social welfare programs, and drug-related crime. From society's perspective, one group gains what the other loses, with a net slippage equal to the administrative costs. These administrative expenses (a small proportion of the value of funds transferred between the two groups) yield no benefit to those who paid for the social welfare programs and no benefits to the recipients: it is simply a dead weight loss in effecting the transfer between the two groups.

In involuntary transfers through crime, the value of resources gained by criminals is roughly equal to the value of resources lost by the victims. One segment of society gains approximately what the other loses. The net slippage in this transfer is the value of property destroyed or damaged in the commission of the crime, a loss to both the criminal and the victim.

G. Perform Discounting

Social intervention strategies may involve long-term and short-term benefits. In economic analysis it is appropriate to adjust benefits accruing over different time periods to reflect their present benefit to society because many of the consequences of the ADM disorders are long lasting, and there is reason to believe that effective interventions yield long-term, as well as, current benefits. The most obvious example is that of premature mortality where alcohol abuse, drug abuse, and mental illness exact substantial tolls from suicide, homicide, motor vehicle crashes, cirrhosis of the liver, and drug overdoses. A rationale for discounting future benefits to the present is . . ."the observation that, all things being equal, people prefer benefits (including health benefits) today rather than at a future time." (U.S. Congress, OTA, 1980a.)

Future benefits are discounted into present values using an appropriate rate of interest. Usually calculations are made with several discount rates: recommended rates are 2.5 percent, 6 percent, and 10 percent.

Even if the ADM disorders do not result in premature death, they may have long-term impacts on employment and productivity. Timely rehabilitation of alcohol and drug abusers may save them from a lifetime of marginal and erratic participation in productive society. Appropriate and timely treatment of addictive and mental disorders may save victims from incapacitation or long-term institutionalization.

With drug abuse, the most feared consequence is death. Almost as important is the fear that an abuser will become addicted to expensive drugs and be propelled into a life of crime. A crime career involves extensive economic cost for society over a long period of time, including damage and destruction of property; injuries to victims; expenditures for police protection, prosecution and adjudication of court cases, incarceration of criminals; and the time that addicted criminals spend incarcerated or in criminal activities rather than socially-approved productive pursuits. Obviously, the economic impacts of drug-related crime are enormous. The estimates presented in this report indicate that over one-third of the economic costs of drug abuse are crime-related.

Public intervention strategies that prevent or interrupt addiction crime careers have current year benefits as well as benefits that accrue over the expected balance of the crime career. For purposes of making cost benefit estimates, the avoided future economic impacts of crime careers are discounted to the year in which the intervention strategy becomes effective.

The future costs of implementing intervention strategies should also be discounted to the present if expenditures stretch into the future. These costs of the intervention strategies are discounted in a manner completely parallel to that for discounting future benefits.

H. Analyze Uncertainties

The impact and effectiveness of ADM intervention strategies are, at best, estimated with some margin for error. The values of important relationships are measured under the best of circumstances with some confidence interval. Other crucial values may have to be inferred, or assumed, based on related studies that do not have ideal information.

For example, some studies use sample surveys. It is well-known that estimates from samples involve potential measurement errors which are reflected by measures such as standard errors of estimates. These measures indicate the probability that true values may be a certain amount greater or less than the estimated value.

Where there is uncertainty in key values for cost benefit analysis, the impact of using alternative (and plausible) values and employing estimates that are considered to be mid-range, low, and high should be explored. This is true for estimates of the benefits, the value of the benefits, and the costs of intervention strategies. Although evaluations of possible intervention strategies should use the most likely estimates, the judgment of decisionmakers should be tempered by the quality and reliability of the available values.

I. Address Ethical Issues

Cost benefit analyses might well identify highly cost-effective interventions, treatments, and techniques that raise ethical, legal, and moral questions. Intervention strategies that involve potential issues of this nature should be carefully scrutinized, and the ethical issues should be laid out for examination by decisionmakers as well as the public.

As the state of medical technology improves and becomes more costly, new ethical issues arise. Some of these issues include access to health care, who shall be treated in the face of scarce health resources, and what level of treatment must be provided. These issues are also relevant to ADM interventions.

Concern with the number of motor vehicle crashes involving teenagers has led many states to raise the legal drinking age limit. Forbidding young adults from drinking alcoholic beverages while otherwise giving them the full rights and responsibilities of majority, however, raises questions of equity. Another policy to fight drunken driving might be to authorize the police to stop and check more drivers on the road at periods of high risk. Several policies and procedures proposed to combat alcohol and drug abuse involve issues of entrapment, invasion of privacy, harrassment, and due process.

There may also be ethical problems with utilization of randomized clinical trials to evaluate alternative treatment regimens. Random assignment of human participants to different types and levels of treatment with unknown levels of effectiveness poses questions for our society.

Such issues must be identified and their merits and demerits debated in parallel with a cost benefit analysis of any proposed public intervention strategy.

J. Interpret Results

The Office of Technology Assessment study points out that cost benefit analyses are used by policymakers and health professionals in deciding what intervention strategies to use, and at what levels. Moreover, the results will be used by the media, and laypeople in the course of debating the merits and demerits of particular programs.

The final responsibility of a professional preparing a cost benefit analysis or cost effectiveness analysis is to provide a careful, clear statement of the findings. The underlying principle of these types of studies should be identical to that for any thorough research study. The procedures and assumptions employed should be documented in sufficient detail to enable an independent party to reproduce the results of the CBA or CEA. The documentation of the results of this and the previous RTI ADM cost-of-illness study has been developed with this principle in mind.

K. Conclusion

The ADM cost-of-illness studies to date are essential inputs to the performance of CBA/CEA analysis and program evaluation. Greater availability of disaggregated data and refinement of methods to estimate indirect and intangible costs should allow continued improvement in computation of ADM cost of illness.

The overall magnitude of ADM cost of illness provides a sense of the relative amount and the nature of resources consumed in the process and treatment of disease. The team of expert consultants who assisted the current study emphasized the need to disaggregate data, to unravel the joint possibility of multiple ADM disorders, and to explore the systemic effects of illness. Available data are certainly not precise enough to account for the overlapping among drug abuse, alcohol abuse, and mental health problems. Consequently, the cost-of-illness estimates (with respect to indirect economic costs) are probably overstated. Better data and econometric technique will help to alleviate this problem in future studies.

Program administration and evaluation can benefit from the improved estimation and analysis of cost of illness. Estimates of COI, however, often are referred to without specificity as to which cost components may be affected by program actions. What costs can be lessened by existing or proposed health programs? A "magic bullet" will not, unfortunately, reduce the costs of ADM illness to zero. The use of CBA/CEA will permit the analysis of alternative actions by a health agency when less than complete eradication of disease is an acceptable and realistic goal.

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V. FUTURE DIRECTIONS IN ADM COST OF ILLNESS STUDIES

Crucial and difficult issues regarding the economic costs of ADM to society remain to be addressed. State-of-the-art estimates have been developed and presented in the preceding sections. In performing the analysis, even more issues have been identified. Many of these were mentioned in appropriate portions of earlier sections of this report.

A final objective of the present study is to highlight methodological issues that should be addressed in order to improve future cost-of-illness estimates. The issues to be highlighted are:

- Overlaps in the ADM populations
- ADM in the workplace
- ADM overlaps in specialty treatment settings
- ADM and other health problems
- Crime
- Fetal alcohol syndrome in adult cohorts.

These are not the only remaining issues for future cost studies of ADM. All of the data and analysis used in these computations can be improved by more intensive and periodical study. These highlighted problems have been studied before, and this report has benefitted from recent advances. They deserve further attention primarily because they are associated with major costs estimated in this study, and the potential for making significant improvements seems high.

A. Overlaps in the ADM Populations

The ADM disorders are often treated as three very separate problems for society although, in fact, they are very closely intertwined. The present study and its predecessor have made separate estimates for the economic costs of alcohol abuse, drug abuse, and mental illness. These have been modeled on cost studies stretching back to 1958 that treated mental illness, alcohol abuse, and drug abuse separately.

The objective of making separate but concurrent cost estimates for each of the ADM disorders required the disentanglements at the overlaps. The data used in these attempts have not been entirely satisfactory for this purpose, making this an important area for improvement in future cost studies.

The most obvious examples where overlaps are important is in the periodic national household surveys of alcohol abuse and drug abuse. These are extremely important data sources about the extent and nature of these two addictive disorders in our society. However, neither the survey on alcohol abuse nor the survey on drug abuse adequately addresses the issue of the abuse of a broad range of substances. The 1979 National Survey on Alcohol Abuse included no questions about abuse of psychoactive substances other than alcoholic beverages.

The 1982 National Household Survey on Drug Abuse included a very small set of questions about alcohol abuse by respondents. However, it has insufficient detail to determine whether an individual might be an alcohol abuser by any of the commonly used criteria.

It has been established in surveys of youth (Rachal, Guess, Hubbard, Maisto, Cavanaugh, Waddell, and Benrud, 1980) that there is a substantial overlap between heavier drinkers and marijuana users. Up to half of the heavier drinkers also use marijuana at least once a week, and a third of the youth who use marijuana more than once a month also are classed as heavier drinkers. Data from the 1982 National Household Survey on Drug Abuse (Miller et al., 1983) indicate that up to 50 percent of young adults that are heavier users of marijuana are heavier drinkers.

In the face of such wide overlaps between young alcohol and drug abuse populations, it is essential to study the incidence, prevalence, and consequences of both disorders in the same populations, and for the same individuals.

There are likely to be equally significant overlaps between other mental illnesses and alcohol and drug abuse although the nature of the overlap is not well understood. The Treatment Outcome Prospective Survey (Rachal et al., 1981) found a high prevalence of symptoms of depression among drug abuse treatment populations. Similarly, Collins and Schlenger (1983) indicated that there is significant overlap between the addictive disorders and other mental disorders in prison populations.

Probably the best hope for identifying and potentially disentangling the overlap between alcohol abuse, drug abuse, and other mental disorders is the epidemiologic catchment area program being sponsored by NIMH (Eaton et al., 1981). Preliminary analysis by Myers et al. (1983) suggests that the presence of multiple ADM disorders is a possibility, since their findings show that the sum of the prevalence rates for a number of distinct mental disorders (including alcohol and drug abuse) is greater than the rate for the presence of any mental disorder. That research has not yet addressed the overlap issue, although it will be possible to indicate the magnitude of the overlap between alcohol and drug abuse, and other mental disorders in the future.

Given the strong indications of overlap between the young alcohol abusing and drug abusing populations, it would also be a significant advance if the national surveys of each of the disorders would collect some baseline information on the other disorders. Ideally, information would also be collected about mental disorders.

B. The Effect of ADM in the Workplace

The largest cost component for each of the separate disorders was lost employment and reduced productivity. From an economic perspective, these are the most significant cost components and, in fact, estimation of this cost component for alcohol and drug abuse was a major effort in this study. Within this general topic there are areas demanding further investigation. The most important of these is reduced productivity due to mental illness.

State-of-the-art econometric estimates of the impact of alcohol and drug abuse on productivity were made in the present study, but only partial estimates could be made for the impact of mental illness in the workforce. The part that could be estimated was for individuals who were institutionalized and for noninstitutionalized persons who indicated that they suffered a complete or partial work disability due to severe emotional disorders or chronic nervous disorders. These cost impacts totaled $22 billion for 1980. These estimates represented only a fraction of the adult population who have been estimated to experience mental disorders during the course of a year. The ECA studies (Myers et al., 1983) found that 12 to 18 percent of the adult population experienced symptoms of a mental disorder over a six-month period (this includes alcohol and drug abuse, which were not estimated separately). The estimates in this study of productivity losses due to mental illness include less then 1 percent of the adult population, a very small proportion of the total who are believed to experience mental disorders.

It is hypothesized that mental illness significantly affects the productivity of its victims, even though they may not seek clinical assistance or acknowledge that they have a problem. This problem can be addressed in the near future as the data sets from the epidemiologic catchment area studies become available for public analysis.

State-of-the-art econometric estimates of the impact of alcohol and drug abuse on worker productivity have been produced as part of the current effort. The research represents the most comprehensive examination made to date. It has been found that particular patterns of alcohol abuse and drug abuse significantly affect the household incomes of ADM victims. This is true even after controlling for other factors known to be significantly related to labor market success.

These state-of-the-art estimates can be improved in several ways. First, slightly different information on labor market participation, earnings, and income should be obtained in future cycles of the NIAAA and NIDA household surveys. Since both Institutes plan to perform future national surveys, this change could be implemented with negligible effort and cost.

Another issue is the difference in the nature and amount of diagnostic information obtained by the respective national surveys. As noted previously, neither survey obtains detailed information about the other addictive disorder. Both surveys obtain relatively complete information about patterns of use and abuse of the respective substances, including current use, and lifetime patterns.

A significant strength of the NIAAA survey is that it collects a great deal of detailed information about many symptoms and potential consequences of alcohol abuse: symptomatic drinking, interpersonal problems, drinking and driving, belligerence, family disputes, and impacts on work.

The survey sponsored by NIDA obtains much less information of this nature about the impacts and consequences of an individual's drug abuse problem on herself/himself, family, friends, and society at large. The 1979 and 1982 national surveys began to investigate some consequences, such as driving and drug abuse and the amotivational syndrome, but there is still a large gap between the quantity and quality of information on tangible impacts obtained by the two national surveys.

For both alcohol and drug abuse, there is a remaining question of whether other factors such as personal values, personality traits, attitudes, and risk-taking behaviors underlie both the substance abuse itself and the tangible consequences such as productivity losses. Factors like these may have preceded substance abuse problems and, in fact, contributed to them. They are probably also determinants of labor market success. Furthermore, attitudes, values, and other behaviors may not change when an individual modifies his or her substance abuse problem. If factors such as these are underlying causes of both substance abuse and level of success in the labor market, then a rigorous study of the impact of substance abuse on labor market success must control for these items to give unbiased estimates of the impact of substance abuse.

These issues will have to be addressed in future studies, since the large scale surveys performed to date have not collected information on substance abuse, employment and income, attitudes, personality, and behaviors which are necessary to make a full study of these relationships.

C. ADM Overlaps in Specialty Treatment Settings

The overlap of alcohol abuse, drug abuse, and mental illness constitutes a problem for estimating the direct treatment costs associated with each disorder. Extensive data are collected and maintained about the utilization of health care treatment in the United States. A major focus of these efforts has been to identify the total magnitude of services provided in the United States and to estimate the distribution of these services among different health care problems. These national data bases have been the foundation for the estimates developed and presented in this study.

Utilization data are usually presented by the health problem and by the initial primary diagnosis made by the health care professional. This practice is not readily compatible with the problem of multiple disorders. Typically, a health care professional makes a primary diagnosis and lists secondary problems as well. Alternatively, professionals may list multiple diagnoses. In practice, when multiple diagnoses are given, the first listed diagnosis becomes the primary diagnosis for reporting and analysis purposes.

While ADM specialty settings may have a particular orientation, specializing in alcohol abuse, drug abuse, or other mental disorders, because of the overlap problem they are likely to be treating people with any or all of the ADM disorders. The convention of using and analyzing data based on primary diagnosis or a first listed diagnosis will not accurately reflect the nature of a patient's problems nor the level of resources required to treat them.

The cost implications of treating multiple ADM disorders should be studied in the future. Improved cost estimates can be produced by identifying services provided to persons with multiple ADM disorders. In addition to analyzing treatment utilization by "primary" and "first-listed" diagnosis, data could be generated showing service delivery to persons with single disorders (with no overlaps) and those with various combinations of disorders (any two of the three ADM disorders, or all at the same time). Making these improvements requires that treatment facilities and clinicians maintain and be able to report data on multiple disorders. In effect, "primary" or "first-listed" diagnoses would have to include "combined" categories.

Dividing the costs for overlapping diagnoses would be a challenge for further analysis. It may be more meaningful to report overlapping costs separately and not artificially assign costs to a single disorder.

D. ADM and Other Health Problems

The overlap problem in general health care settings is identical to that in the ADM speciality sector. An additional factor in the general health care sector is that alcohol and drug abuse are each related to other health care problems. The relationship of alcohol abuse to other health care problems receives intense scrutiny in the medical profession, and results are regularly summarized in the NIAAA reports on alcohol and health. Alcohol has been indicated as an underlying cause of a number of health problems that also have other causes. Ongoing epidemiological research includes a thorough analysis of these data to discover the proportions of other illness that might be attributable to alcohol abuse. Cost attributions for these other illnesses can be made to alcohol abuse using those established relationships. Alcohol abuse also has cost implications when it exacerbates health problems that it does not necessarily cause. Some preliminary studies on this problem using data maintained by health care insurance companies have compared health care utilization in households where some member has received treatment for alcohol abuse with other households. Further insights into the problems associated with alcohol abuse will be gained from this avenue of research.

Important research into the relationship between alcohol abuse as a complicating factor in other illnesses is being performed by NIAAA. Initial findings indicate that alcohol-specific illnesses such as liver cirrhosis, alcoholic psychosis, and alcoholism are secondary causes of death for about two-thirds again as many mortalities as they are primary causes. Some proportion of these mortalities should be attributable to alcohol abuse. Analogously, where alcohol abuse complicates health care treatment for other illnesses, some proportion of those costs should be identified and included in future cost studies.

Knowledge about drug abuse and health problems is not as advanced as that for alcohol abuse. Drug overdoses are a well-known consequence of drug abuse. Further health problems involved with abusing various drugs are hypothesized but require further research, documentation, and summarization.

The health effects of the most widely abused illicit substance--marijuana--were recently reviewed by a committee of the Institute of Medicine (Institute of Medicine, 1982). Conclusions reached in that study were that, while there are "a broad range of psychological and biological effects," it is not known how extensive the health problems is or "how serious this risk may be." The state of knowledge regarding biological effects of other illicit substances is comparable. While effects of various types have been observed, there is a lack of epidemiological evidence about their extent and severity.

Overlaps of mental illness with substance abuse should be dealt with in the future. It is not known in which direction overlaps may have biased the current cost estimates for mental illness.

E. Crime and ADM

The strong relationship between ADM and crime has received intense study over the last ten years and is a continuing topic of debate. Significant improvements have been made in the course of this study in estimating the economic costs associated with ADM. Despite the improvements in data and methodology, important issues remain open for continued analysis.

Mental illness has been linked to violent crime. A study of a prison population by Collins and Schlenger (1983) indicated that nearly 70 percent of the inmates exhibited some disorder, ranging from alcohol and drug abuse, to antisocial behavior disorder or other mental disorders. Nonetheless, the difficult question remaining to be answered is what proportion of various types of crimes is attributable to mental illness? Only when this question is answered can costs be assigned to mental illness and crime.

The drug abuse/crime relationship has been analyzed by interviewing prison inmates, studying the drug usage patterns of arrestees, analyzing the behavior of drug abuse treatment populations, and conducting intense ethnographic studies of small groups of drug addicts on the streets. All of these studies indicate a high involvement of drug abusers with criminal activities. However, most of these studies have focused on a limited and not necessarily representative segments of either the drug abuse population or the criminal population. Both of these populations are extremely difficult to identify, and members have little incentive to give reliable data on their antisocial patterns of behavior. Studies of drug abuse should carefully examine the implications of their findings for crime and vice-versa, and the results should be compared with other sources of data such as the victimization surveys performed by the Bureau of Justice Statistics and the regularly compiled data from the Uniform Crime Reports. To establish reliability and validity of the microlevel studies of select populations, the implications from the study groups must be extrapolated to a regional or national level and compared for consistency with aggregate information on the value and number of crimes committed.

F. Fetal Alcohol Syndrome in Adult Cohorts

Economic implications of the fetal alcohol syndrome have been explored for the first time in this study, building on and modifying the cost information collected by Russell (1980). The estimated costs for FAS are over 2 percent of the total value for all of alcohol abuse. Although all of the estimates for FAS are state-of-the-art and subject to revision given further study, there are several aspects of FAS that are most critical for these types of cost of illness studies.

The subject of increasing concern since the early 1970s, research on incidence and prevalence of the fetal alcohol syndrome has focused primarily on new birth cohorts. FAS, however, effects symptomatic individuals throughout life, imposing costs on them, their families, and on society. We must ask whether it is possible to identify FAS victims in older birth cohorts, including juveniles, teens, and adults. Since FAS is identified by a set of permanent defects and deformities, it is hypothesized that prevalence estimates can be made for these older cohorts.

The single aspect of the syndrome that is most economically significant is mental deficiency. Research indicates that all FAS victims experience some kind of mental deficiency, ranging from minimal brain dysfunction to severe/profound mental retardation. To make more precise estimates of FAS costs, more reliable estimates must be made of the prevalence of various degrees of mental deficiency. Moreover, reliable information is needed about the level of functionality that FAS victims may achieve in society. The severely and profoundly retarded victims are likely to have a minimal capability to function in society and would, therefore, be institutionalized. Lower levels of mental deficiency should involve greater capability to function in society. It must be ascertained whether FAS victims can do skilled or unskilled work, and whether they must work in a sheltered workshop environment.

G. Conclusions

The research needs identified in this section have strong implications for cost studies of alcohol and drug abuse and mental illness. Rigorous analysis of these issues could yield higher or lower new estimates. The overlap problem may require costs assigned to one disorder to be split with or attributed to another disorder. Changes may occur as knowledge about the very complicated ADM disorders expands.

The issues identified here represent a short but important list of research needs for improving future cost of ADM studies:

- Overlaps in the ADM populations
- ADM in the workplace
- ADM overlaps in specialty treatment settings
- ADM and other health problems
- Crime
- Fetal alcohol syndrome in adult cohorts.

Major advances have been made in the present study on some of these topics. Yet, this work has served more to define how much more must be done than to yield definitive results.

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Appendix

A. THE IMPACT OF ALCOHOL AND DRUG ABUSE ON PRODUCTIVITY IN THE WORKFORCE

A. THE IMPACT OF ALCOHOL AND DRUG ABUSE ON PRODUCTIVITY IN THE WORKFORCE

A. Introduction

Although alcohol and drug abuse in the workplace have been topics of great concern, they have received surprisingly little study. Some limited studies have examined the extent of problem drinking in the workplace, and others have made rough estimates of the costs to a company of having an alcoholic employee. The major cost-of-illness studies are reviewed here for their treatment of lost productivity. This is followed by a discussion of some recent work that has examined how health affects labor market success of individuals.

This section examines the impact of alcohol and drug abuse on the labor market success of individuals. Individuals with a substance abuse problem, it is contended, are less productive than other people with the same characteristics because of impaired social, mental, and physical functioning. Alcohol or drug abuse may cause problems outside the workplace that, nonetheless, affect functioning on the job. The cause and effect relationships among alcohol and drug abuse, the work environment, and other social factors are not clear. Stress and depression can affect an individual's productivity and may also contribute to a substance abuse problem. Too, the attitudes, values, and personality traits which underlie substance abuse behaviors as well as all others should be incorporated in future analysis; however, it was not possible with the data sets presently available.

B. Cost of Illness Studies and Productivity

In studies of ADM, the largest cost has been losses of productivity in the workforce. In the 1981 study, over 50 percent of the economic cost of alcohol abuse was due to reduced productivity and lost employment; for mental illness, these components made up nearly 35 percent of the total cost, while for drug abuse they made up about 18 percent.

Other studies (Rice, 1966) have found that fully 25 percent of the economic costs for all illnesses was due to persons who were completely unable to work for all or any portion of the year. A more recent report by Cooper and Rice (1976) estimated that in 1972 lost work time due to illness made up 22 percent of the total economic costs for all illnesses. The most recent study of all illnesses (Paringer and Berk, 1977) also estimated that lost work time due to illness was 24 percent of the total economic costs of all illnesses.

Each of these studies also examined mental disorders--psychoses, neuroses, alcohol psychoses and dependence, drug dependence, and mental retardation. The three studies estimated that 64 percent, 45 percent, and 46 percent, respectively, of total costs were due to the lost work production of those experiencing mental disorders.

The indirect morbidity costs estimated in each of those studies only included lost productive time during which the individual could not perform his or her primary activity due to confinement in the home, in a short-term health facility such as a general hospital, or in a long-term residential facility such as a psychiatric hospital or nursing home.

More recent work by health and labor economists has expanded the concept of economic productivity losses. Studies of employment and labor market success have used measures of health status as an important determinant of factors such as labor force participation, hours and weeks worked per year, and wage and salary rates. Examining all of these facets, Luft (1975) compared persons reporting long-term disability to the non-disabled and estimated that the long-term disabled lost $23 billion in aggregate earnings in 1966. This estimate included components for nonparticipation in the labor force, limitations in the weeks and hours worked, and impacts on wage rates. Luft concluded that long-term disability affected all aspects of labor market success.

Luft's study was similar to that of prior cost-of-illness studies in that it included the value of time when individuals could not work due to confinement at home and in hospitals. The unique contribution of this study was an analysis of earnings lost due to the reduced productivity of disabled individuals. Luft concluded that productivity losses due to long-term disability were 6.2 percent of total adult earnings in 1966. Comparing the disabled to the nondisabled, he found that white males had a reduction in annual earnings of 21 percent, black males a reduction of 24 percent, white females a reduction of 36 percent, and black females a reduction of 25 percent (see table A-1).

Table A-1

Impact of Health on Labor Market Success in Select Studies

		Impact of Health (Proportional Change)			
Study Population	Health Problem	Annual Earnings	Wage Rate	Unemployment Rate	Labor Force Participation
Luft (1975)					
Black Males	Long-term Disability	-24.3	-10.3	+50.2	-28.8
Black Females	"	-25.1	+3.8	+34.3	-30.7
White Males	"	-21.3	-11.6	+69.9	-18.6
White Females	"	-35.8	-9.8	+31.7	-30.2
Bartel and Taubman (1979)					
White Males Age (46-56)	First Diagnosed Mental Disorder				
	1967-1973	-23.9	N.A.	N.A.	N.A.
	1962-1967	-22.0	-16.4	+1.3%	-5.0%
	1955-1961	-13.3	N.A.	N.A.	N.A.
	1948-1957	0.0	N.A.	N.A.	N.A.
McManus (1978)					
White Male Workers	Disabled 1 Year	N.A.	-5.2	N.A.	N.A.
	Disabled 2 Years	N.A.	-9.2	N.A.	N.A.
	Disabled 3 Years	N.A.	-12.2	N.A.	N.A.
	Disabled 4 Years	N.A.	-14.1	N.A.	N.A.
	Disabled 5 Years	N.A.	-15.1	N.A.	N.A.
Berry and Boland (1977)					
Households with an Adult Male	Problem Drinker	-18.4[1]	N.A.	N.A.	N.A.

[1] Household income. No other aspects were examined.

A study by McManus (1978) examined the impact of long-term disability on earnings. His primary focus was the impact of disability on the wage rates of those who remained in the workforce. The results indicate that a person disabled for five years but still in the workforce has a wage rate 17 percent below that which he would have received otherwise.

A more recent report by Bartel and Taubman (1979) made a significant advance over prior studies by using incidence data for specific diseases as diagnosed by health providers. Although the study had excellent data both on employment and health, it was somewhat limited by being drawn from the National Academy of Science twin panel data set. Because the study population was only white veteran male twins born in the continental United States between 1917 and 1927, limited generalizations can be made from the study findings.

Nonetheless, Bartel and Taubman made some important findings with respect to the impact of mental illness on earnings. They found that individuals who had a diagnosis of a psychosis or neurosis (excluding alcoholism and drug dependence) experienced significant negative impacts on their earnings, wage rate, the amount of time worked per week, and their probability of being in the labor force. An initial diagnosis of mental illness five years prior to the survey reduced earnings by 24 percent. A first diagnosis five to ten years prior to the survey affected earnings negatively by 22 percent, while a diagnosis ten to fifteen years previously reduced earnings by 12 percent. Diagnoses more than fifteen years prior to the date of survey had no significant impact on earnings in the survey year. This finding, as in the studies by Luft and McManus, controlled for a number of sociodemographic factors which are known to be important determinants of labor market success. These included education, age, marital status, and occupation or socioeconomic status.

Bartel and Taubman (1979) estimated that $1.7 billion in earnings were lost due to psychoses/neuroses in 1972. They cautioned that their specific results had limitations in their generalizability due to the nature of the sample. Nonetheless, the robustness of their results indicates that mental illness affects individuals' wage rates as well as their presence in the workforce or on the job at a particular time. Furthermore, it suggests that the impact may be very long lasting.

The first study to include estimates of the value of reduced productivity attributable to an ADM illness was Berry and Boland (1977). Using a 1968 national household survey on alcohol abuse, they compared the income of households with noninstitutionalized males, aged 21 to 59. The income of households with a male problem drinker (defined by having a large number of alcohol-related consequences) was 18.4 percent lower than the income of households without a problem drinker. When the problem drinker was 21-29 years old, the difference was 18 percent; 30-39, the difference was 17 percent; 40-49, the difference was 19 percent; and 50-59, the difference was 10 percent.

In comparing households with and without male alcohol abusers, the Berry and Boland analysis was not able to control for factors such as education, family structure, occupation or socioeconomic status, and age within the ten-year brackets. Consequently, they chose to adjust the 18.4 percent difference down to 14 percent based on a finding in Luft (1975) that 23.9 percent of the difference in annual earnings between disabled white males and healthy white males was due to factors such as age, education, marital status, and a few other factors.

C. Approach of this Study

This study has developed estimates of the economic impact of alcohol abuse and drug abuse on the productivity of the workforce. Analyses in this study were performed in a manner analogous to those by Luft (1975), Bartel and Taubman (1979), McMannus (1978) and Berry and Boland (1977) and make a significant improvement over the results found by Berry and Boland. In this study it has been possible to control for a number of factors that are known to affect labor market success, while examining the impact of problem drinking and drug abuse.

The approach taken for both alcohol abuse and drug abuse was to compare the income for households with a substance abuser present with that of households with no abuser present. This comparison was performed while controlling for characteristics of the respondent in the household, including age, education, sex, marital status, and occupation and for the presence of children in the household. This comparison was performed by using multivariate regression analysis. For alcohol abuse, this analysis was performed using data from the 1979 national household survey on alcohol abuse sponsored by NIAAA (Clark and Midanik, 1982). The analysis of drug abuse was performed using data from the 1982 national household survey sponsored by NIDA (Miller et al., 1983). Both data sets were used because neither had information about respondents' use of both alcohol and illegal drugs. The 1982 national drug abuse survey asked a few questions about current alcohol consumption but none about lifetime patterns and consequences of drinking. Similarly, the 1979 national alcohol abuse survey obtained no information at all about abuse (past or present) of illegal drugs but had very detailed information about alcohol use and consequences of drinking.

The design of both national surveys presented specific limitations on this analysis. First of all, only one adult was interviewed per household. All information obtained through the questionnaire is specific to that individual. The 1979 survey asks about patterns of alcohol consumption, life events, and impacts of alcoholic beverages on the respondent's functioning at home, in public, and on the job. Given this, annual earnings or wage rate should also be specific for the individual, but both surveys asked for only the annual household income with source of income unspecified.

Household income frequently includes the earnings of more than one individual and can include benefits received from social welfare programs (such as unemployment insurance, aid to families with dependent children, disability payments) as well as income from savings accounts, stocks, bonds, or trust funds (i.e., unearned income).

Berry and Boland were also forced to use household income. Their estimates are comparable to and limited in the same way as the estimates developed for this study. Thus, analysis of the impact of income on alcohol abuse must proceed very carefully.

In this study it has not been possible to control for unearned income, but it has been possible to control at least partially for the earnings of other individuals within the household. The two surveys do obtain information about the marital status of the respondent and the presence of children under 12 within the household. Some assumptions can be made about income based on the presence of a spouse and young children.

1. Productivity Losses and Alcohol Abuse

The first task in the analysis was to identify the sociodemographic characteristics that are known to affect labor market success.

These included education, age (coded as number of years since final year in school), race, sex, marital status, presence of children under the age of 12 in the household and, if the survey respondent was in the labor force, occupation (see table A-2 for variable definitions). Using the 1979 survey, a regression of these variables on household income found the relationship to be very significant, with an F value of 36.6, and an R^2 of .392. This basic set of sociodemographic factors was able to explain 39.2 percent of the variance in household income.

The second task in the analysis was to examine the impact of drinking patterns and problem drinking on income after controlling for all previous factors. The simplest approach for this analysis would be a simple comparison of sociodemographically similar households with and without alcoholics or problem drinkers. Berry and Boland used this approach by applying a problem drinking index which Cahalan (1970) had developed for the specific survey that they used. That particular index aggregated a number of different kinds of problems which obscures numerous facets of problem drinking. The procedure in this study was to examine the impact of specific problem drinking symptoms on household income while controlling for the sociodemographic variables specified above. From the many problem drinking symptoms obtained in the survey, only those on which ten or more of the 1,772 survey respondents answered affirmatively were used. It was judged that any item with ten or fewer positive responses, no matter how important an indicator, would most likely fail to be significant in statistical testing. Therefore, only a subset of all symptoms was examined.

Extensive testing was performed on many different kinds of problem drinking symptoms (see table A-3 for a partial list of variables tested). Symptoms were included in the regression model along with the controlling sociodemographic factors to indicate whether or not they were significantly related to household income. Although virtually all of the indicators tested had a negative relationship with household income, the vast majority did not have a statistically significant relationship to household income when sociodemographic factors were controlled. However, four specific problem symptoms were identified as having the hypothesized negative affect on household earnings and being statistically significant. However, these four symptoms--binge drinking, tardiness or absence from work because of a hangover, marital problems attributed to drinking, and arrests for driving

Table A-2

Base Variable Definitions for Final Regressions on Alcohol Abuse

ED1-ED6:	Dummy variables (0/1) indicating education level attained
ED1:	Less than 7 years
ED2:	Some junior high school
ED3:	Some high school
ED4:	High school graduate
ED5:	Some college or university
ED6:	College graduate or beyond
POTEXP:	Computed as (Age minus Number of years of Education minus 5)
POTEXP2:	Square term of POTEXP
NRACE:	Race: White defined as zero, other defined as one
PROF-LABORER:	Dummy variables (0/1) indicating type of employment
PROF:	Business executive or professional
MAD:	Manager and administrator
SALES:	Retail or office worker
CLER:	Clerical
CRAFT:	Craftsman (includes men enlisted in armed forces)
LABFM:	Laborers and farmers
OPER:	Operators
SERV:	Service
KIDS:	Dummy variable (0/1) indicating presence of children under the age of 12 in the household
D0-D3:	Variables created to represent the sex and marital status of individuals in the labor force
D0:	Single males
D1:	Married males
D2:	Single females
D3:	Married females
QF:	Quantity/frequency variable measuring average daily alcohol consumption
QFSQ:	Square term of QF
PD:	Dummy variable (0/1) created for persons with positive responses to one or more of the following components of problem drinking
HANGOVER:	Ever late to or miss work because of a hangover?
MARPROBS:	Have problems with spouse or ex-spouse in the past year because of drinking?
DRIVE:	Ever arrested for driving while under the influence of alcohol?
BINGEYR:	Have you gone on a drinking binge in the last 12 months?

Source: Research Triangle Institute.

Table A-3

Variables Tested to Create a Measure of Drinking Problems

Hangover:	Ever missed or been late to work because of a hangover?
MarProbs:	Had problems with spouse or ex-spouse because of drinking?
Drive:	Ever arrested for driving while under the influence of alcohol?
Bingeyr:	Have you gone on a drinking binge in the last 12 months?
Symptoms:	Aggregate of number of drinking symptoms in the past year
Freqhigh:	Frequency variable created measuring how often drunk in the past year
Forget:	Query of why drink: to forget everything?
Worries:	Query of why drink: to forget worries?
Cheerup:	Query of why drink: because helps to cheer you up?
Tense:	Query of why drink: when tense and nervous?
Accident:	Ever been involved in an accident after drinking?
Arrest:	Ever been arrested in connection with drinking?
Drkdrive:	Ever been a passenger while driver was drunk?
Healthprob:	Aggregate tested, along with each individual problem

Source: Clark and Midanik, 1982.

while under the influence of alcohol--are strong indicators of problem drinking. The four significant symptoms were then combined into a single indicator of the presence of one or more of the four symptoms.

The survey included extensive information of drinking patterns as well as drinking impacts. Drinking patterns were examined for their relationship to household income. Tests were run on number of times drunk, the number of times high but not drunk in the last year, a quantity-frequency measure representing average daily consumption of absolute alcohol over the past year, and a quantity-frequency measure representing absolute daily consumption of alcohol during the past month. The first three pattern variables were not found to be significant. The last variable, representing the current level of consumption of alcohol, had a very strong relationship to household income, even when controlling for the sociodemographic characteristics.

Fully 10 percent of labor force participants in the 1979 national survey had one or more of the four problem drinking symptoms (table A-4). This prevalence rate compares very closely to prior estimates of problem drinking and alcohol abuse. In 1979, young males had the highest prevalence of drinking problem symptoms. In general, males had higher rates than females. Females in the 20-24 year age group had the highest rate among women.

Recall that in the report by Clark and Midanik (1982) on the 1979 national survey, 10 percent of the adult population showed some symptom of loss of control which indicated alcohol dependence. The rate for males was 15 percent, and for females it was 6 percent (see table III-13). Similarly, 5 percent of the adult population (7 percent of males and 3 percent of females) experienced one or more social consequences that they directly attributed to alcohol abuse. There is some overlap between the estimates for loss of control or dependence and that for social consequences.

From Clark and Midanik's data it is clear that problem drinking symptoms are highest between ages 18 and 30. The lowest rates for both males and females are for those aged 60 and above. This age distribution of problem drinking symptoms means that the prevalence would be even higher for adults between ages 30 and 60, the years in which they are most likely to be in the labor force. The study by Berry and Boland (1977) found 17.5 percent of households with a male aged 21-59 had a problem drinker

Table A-4

Prevalence in the Workforce of Problem Drinking Symptoms and Daily Use (Ever) of Marijuana by Age and Sex (in Percentages)

Age	Problem Drinking Indicator†		Daily Use (Ever) of Marijuana	
	Male	Female	Male	Female
18-19	16.4	5.7	18.9	14.3
20-24	21.0	15.4	21.8	11.0
25-34	18.5	4.5	12.5	4.5
35-44	11.9	8.2	0.0	0.0
45-54	13.3	5.6	0.0	0.0
55-64	6.0	0.8	0.0	0.0

Sources: Calculations from the 1979 Household Survey on alcohol abuse funded by NIAAA, and the 1982 Household Survey on drug abuse funded by NIDA.

†See PD variable definition, table A-2, for the definition of "Problem Drinking Symptoms."

present (see table A-5). Assorted other studies have indicated 5 percent of those in the workforce were alcoholics and, perhaps, another 5 percent were problem drinkers. Table A-5 shows that many of these same studies also estimated the impact of alcoholism or problem drinking on worker productivity as being between 20 and 30 percent. The exception to this was an analysis of Berry and Boland, whose initial estimate was an impairment level of 18.4 percent. However, they made another adjustment based on completely unrelated data which dropped their estimate of impairment to 14.0 percent. As stated previously, the estimates by Berry and Boland did not take into account important determinants of the labor market success which this analysis has been able to utilize.

The regression analysis of the impact of problem drinking on household income is summarized in table A-6. Only the results for drinking-related variables are presented there. The dependent variable in the relationship was the natural logarithm of household income. The independent variables were the items specified in table A-2 (referred to as the "base" variables) and the drinking related variables. In a regression using a logged dependent variable, coefficients of unlogged independent variables may be interpreted as the proportional impact of a 1 unit increase in that variable when the coefficients are relatively small (between +.10 and -.10). At values beyond that, the proportional impact is obtained by taking the antilog of the coefficient and then subtracting "1." Positive coefficients represent an increase, and negative coefficients reflect a negative effect.

The analysis was performed on three somewhat different groups. The first was individuals in the labor market (table A-6, lines 1, 2, and 5), those employed as well as those unemployed at the time of the survey. The second group was the total population (line 3). The third group was respondents that were employed (line 4). The first group is generally considered to be the best for addressing the current question because an individual must be working or looking for a job in order for substance abuse to affect his or her earnings. Included in this group are those who, though currently not working, have held a job recently and can reasonably be considered part of the labor force. Looking at the problem of lost earnings more comprehensively, consideration should be given to those who have disabling ADM problems which have either caused them to be frequently terminated from jobs and, therefore, to be unemployed for various periods or caused them to

Table A-5

Prevalence and/or Reduced Productivity of Alcohol Abusers and Alcoholics as Estimated by Various Studies

Study	Estimated Prevalence	Defined Population	Reduced Productivity	Defined Population
Vischi et al. (1980)	10 million	Adult problem drinkers	not estimated	n/a
Cahalan et al. (1969, 1970, and unpublished data)	9 percent 16 percent	Adult problem drinkers Male adult problem drinkers	18.4 percent	Reduced income of households with male adult problem drinkers present (unadjusted for age, education, and family structure).
Keller (1971)	5 percent 5 percent	Labor force alcoholics Labor force other problem drinkers	not estimated	n/a
National Council on Alcoholism (1971)	4.6 percent	3.6 million untreated alcoholic employees out of 79 million persons employed. Secondary data.	25 percent	Alcoholic employees - losses to company due to lost production and other costs. Secondary data.
Winslow et al. (1966)	not estimated	n/a	23 percent	Suspected problem drinkers compared to problem-free employees. Primary data with matched samples.
Swint and Nelson (1977)	5 percent	Alcoholics in work force. Secondary sources.	20-30 percent	Alcoholic employees. Secondary sources.
Schramm (1974)	5 percent	Alcoholics in work force. Assumption based on secondary sorces.	25-50 percent	Alcoholic employees. Assumed level of reduced productivity.

(continued)

Study	Estimated Prevalence	Defined Population	Reduced Productivity	Defined Population
Berry et al. (1977)	17.5 percent of households with a male aged 21 to 59	Male adult problem drinkers Based on Social Research Group's national probability sample of households (Cahalan, unpublished data)	14.0 percent	Reduced income of households with male adult problem drinkers present (adjusted for age, education, and family structure).
Mannello and Seaman (1979)	19 percent	Problem drinking railway workers (using Cahalan et al. 1970)	29 percent	By evaluation of supervisors: problem drinkers versus average employees. Supervisors diagnosed likely problem drinkers. Not related to prevalence estimates.
	12 percent	(using independent methodology)		
	15 percent	(by Program Directors standards)		
	21 percent	(combination 1 and 3)		
	9.3 percent	(workers' self-evaluations)		
VonWiegand (1976)	6 percent	Alcoholic employees. Secondary sources.	27.7 percent	Alcoholic employees. Cost due to lower productivity, absenteeism, accidents, etc. Secondary sources.
Swint and Nelson (1977)	5 percent	Alcoholics in work force Secondary sources.	20-30 percent	Lower productivity of alcoholics (absenteeism and work output). Secondary sources.

Source: Cruze et al., 1981.

Table A-6

Regression Results for Analysis of Household Income[1] and Problem Drinking (By Percentage)

Dependent Variable	Problem Drinking Indicator	Quantity/ Frequency	Quantity/ Frequency Squared	F	R^2
1. Household Income of Labor Force Participants	-.138 (-2.33)**	-	-	35.1	.396
2. Household Income of Labor Force Participants	-.2357 (-3.65)†	.229 (3.66)†	-.0479 (-2.74)†	32.2	.402
3. Household Income of Total Population	-.2367 (-3.82)†	.261 (5.59)†	-.051 (-4.71)†	67.0	.484
4. Household Income of Employed Persons	-.282 (-4.26)†	.230 (3.12)†	-.0429 (-1.77)*	27.1	.38
5. Household Income of Labor Force Participants	-	.169 (2.78)†	-.041 (-2.36)*	32.7	.394

[1] This dependent variable is entered in natural log form.

†Significant at the .01 level.

**Significant at the .05 level.

*Significant at the .10 level.

permanently leave the labor force. Data sets to answer these last questions are not available.

Table A-6 is read from left to right. Lines 1 through 5 each summarize a regression. The title next to the number defines the dependent variable for the regression and the population included in the regression. In the table column entitled "Problem Drinking Indicator" is the coefficient in that regression. Under Quantity/Frequency and Quantity/Frequency Squared are coefficients in those regressions. The "F" and "R^2" values indicate the overall strength of the relationship between the dependent variable, and the "base" and drinking variables. The base variables and their coefficients are not shown here in order to focus on the most important relationships between income and the drinking variables.

The results in table A-6 indicate that problem drinkers identified through our analysis were consistently less successful in the labor market than non-problem drinkers with the same characteristics. The initial comparison of household income between the two groups (on line 1) indicates that the presence of one of the four problem drinking symptoms reduced household income by about 13 percent. This result was statistically significant. When variables representing current consumption of alcohol were included (line 2), the impact of problem drinking was found to be much greater: a 21 percent decrement was experienced by the problem drinking households. Furthermore, the (line 2) relationship including current drinking variables indicated that where an individual consumed more than an average of 2.4 absolute ounces of alcohol daily, income was negatively affected whether or not they had any problem drinking symptoms.

When the impact of level of current drinking was assessed without the problem drinking indicator in the regression (line 5), the negative effect of high levels of drinking was still found. At consumption levels beyond approximately 2 absolute ounces of alcohol daily, income fell as consumption rose.

The third and fourth lines have results for the total population and employed persons, respectively. Their results are similar to the relationship for labor force participants. In fact, it was found that problem drinking had an even larger impact on employed persons than it did for the total labor force. This suggests that problem drinkers were less likely to be unemployed than others, but that the hourly wage decrement was greater than expected.

These findings are consistent with previous studies of the impact of alcoholism and problem drinking in the workplace. The prevalence rate, based on the four significant problem drinking symptoms, and the level of reduced productivity are also similar to those estimated in prior studies (table A-5). Moreover, the estimates of the size of the impact of problem drinking are comparable to those cited earlier for long-term disability (Luft, 1975) and for psychoses and neuroses (Bartel and Taubman, 1979). The fact that the current estimates are the same order of magnitude as prior similar estimates adds credibility to the current results.

Productivity losses attributable to problem drinking in 1980 are presented in chapter III. The computations were made by multiplying the number of individuals in the labor force for each age and sex group by the prevalence rate for our problem drinking indicator. This product was multiplied by the expected productivity (labor market plus household) of that group and, finally, by the impairment rate of 21 percent. Separate computations were made for men and women in the labor force, and for those whose primary occupation was homemaking. Summing the losses across all groups indicated reduced productivity attributable to alcohol abuse of $57.7 billion in 1980. Of this total, $44 billion was due to problem drinking by males, and $13.7 billion was due to problem drinking by females.

These estimates are adjusted downward to $49.8 billion in the final report because many young problem drinkers are also drug abusers--that is, because of double counting. This issue is addressed at the end of this section.

2. Productivity Losses Due to Drug Abuse

A similar analysis has been performed to examine the impact of abuse of marijuana on the workforce. The 1982 national household survey on drug abuse focused on patterns of use and abuse of psychoactive substances including marijuana, cocaine, heroin, hallucinogens, and a variety of stimulants, sedatives, and analgesics. Questions on income and sociodemographic factors, however, were almost identical to those in the 1979 alcohol abuse survey.

Like the study on alcohol abuse, the drug abuse study examined patterns of current and lifetime consumption, and patterns of use for a number of different psychoactive substances. The drug abuse study, unlike the study on alcohol abuse, obtained no information about untoward events they might have been due to abuse of drugs. Questions were not asked about areas in which abuse of alcohol is known to have an impact such as symptomatic drug consumption, interpersonal problems, difficulties in the household, legal entanglements, or problems on the job. By analogy, it would be predicted that drug abuse has impacts in the same areas as alcohol abuse, but this has not been examined by any of the national surveys on drug abuse.

Although the national surveys on alcohol abuse and drug abuse had similar sociodemographic information, in fact, the income information in the drug abuse survey was of poor quality for analysis such as this. In both surveys, the income data were obtained in ranges. The alcohol abuse survey had ranges with $5,000 intervals with a total of 11 categories. In the drug abuse survey, income data were in 7 categories, with some intervals as broad as $10,000. Grouping the income data makes it more difficult to obtain precise and statistically significant estimates for relationships.

The prevalence rates of ever using marijuana daily by labor force participants are presented in table A-4. For males age 18-24, this prevalence was about 20 percent. Males aged 25-34 had a lower rate, 12.5 percent. Older males had an effective rate of "0". Rates for females were somewhat lower, although they were greater than 10 percent for women aged 18-24.

Multivariate regression analyses were completed for single males in the labor force, married males in the labor force, all males grouped together, single females in the labor force, married females in the labor force, all females together, and all labor force participants. The sociodemographic control variables (listed in table A-7) were similar to those used for the analysis of alcohol abuse.

The statistical analysis of the impact of consumption of drugs other than marijuana yielded no significant results relating abuse of the drugs to household income. Perhaps this should have been anticipated. First of all, heavy drug users may have life styles that make them unlikely to be captured in household surveys. Second, prevalence of drug use other than marijuana is much lower than that for marijuana. The failure to find relationships was true for variables reflecting recency and frequency of

Table A-7

Base Variable Definitions
Final Regressions on Marijuana Use

ED1-ED6:	Dummy variables (0/1) indicating education level attained
ED1:	No schooling
ED2:	Elementary school--8th grade or less
ED3:	Some high school
ED4:	High school graduate
ED5:	Vocational/technical school beyond high school
ED6:	Some college
ED7:	College graduate or beyond
POTEXP:	Computed as (Age minus Number of years of Education minus 5)
POTEXP2:	Square term of POTEXP
NRACE:	Race: White defined as zero, others defined as one
PROF-LABORER:	Dummy variables (0/1) indicating type of employment
PROF:	Business executive or professional
MAD:	Manager and similar
SALES:	Retail or office worker
CRAFT:	Craftsman, foreman, skilled worker
SERVICE:	Service workers
LABORER:	Laborer
KIDS:	Dummy variable (0/1) indicating presence of children under the age of 12 in the household
D0-D3:	Variables created to represent the sex and marital status of individuals in the labor force
D0:	Single males
D1:	Married males
D2:	Single females
D3:	Married females
DAILYMJ:	Dummy variable (0/1) created for persons who had ever smoked marijuana daily for a month
MONTHMJ:	Frequency variable calculated as the number of marijuana cigarettes smoked per day times the number of different days during the past 30 having smoked marijuana
MONTHMJ2:	Square term of MONTHMJ

Source: Research Triangle Institute.

current consumption, lifetime consumption of the substances, and abuse of substances in combination with alcohol or other drugs. Unfortunately, this was the extent of information obtained about consumption of each of the substances.

Table A-8 is constructed and reads identically to table A-6. Again, all appropriate "base" variables were used in the regression analyses reported in each line. (The variables and their coefficients are not printed here only to keep the presentation clear.) Analysis revealed a large and significant impact on current household income if an individual ever smoked marijuana daily for a period of at least one month. Other variables reflecting current use patterns and lifetime use were not statistically significant. Some variables measuring use had the predicted negative, although insignificant, impacts on household income; some did not; and others indicated positive, though still insignificant, impacts.

The multiple regression results for labor force participants and the total population and employed persons are summarized in table A-8. The predicted impacts of ever using marijuana daily were substantial. In the analysis of household income of labor force participants (line 1), ever using marijuana daily had a highly significant coefficient of -.3276. This translated into a negative impact of 27.9 percent. Examining two alternative specifications, even larger impacts of ever using marijuana daily were indicated. The coefficient in the regression of household income of the total population (line 3) was -.397, a statistically significant 33 percent deficit. The impact is even greater when examining the household income of employed persons (line 4): the coefficient of -.516 translates into a reduction of income of 40 percent.

These results have been used to calculate the reduced productivity due to daily marijuana use. Once again, the prevalence rates of ever using marijuana daily in the labor force by age and sex groups (see table A-4) were applied to the number of persons in the labor force from those groups that experienced reduced productivity. This number was multiplied by the expected productivity (in the workplace and in the household) and then by the impairment rate (27.9 percent) to produce the final value. The loss due to marijuana abuse was estimated as $34.2 billion for 1980.

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Table A-8

Regression Results for Analysis of Labor Market Behavior and Marijuana Abuse (by Percentages)

Dependent Variable	Daily Use Ever	Current Use	Current Use	F	R^2
1. Household Income of Labor Force Participants[1]	-.3276 (-2.83)†	-	-	9.6	.066
2. Household Income of Labor Force Participants[1]	-.401 (-3.13)†	.0047 (1.13)	-.00001 (-.70)	8.8	.067
3. Household Income of Total Population	-.397 (-2.61)†	.005 (1.10)	-.00001 (0.53)	11.0	.061
4. Household Income of Employed Persons[1]	-.516 (-4.55)†	.0085 (2.09)*	-.000027 (1.61)	8.5	.07

[1] This dependent variable is entered in natural log form.

†Significant at the .01 level.

*Significant at the .05 level.

As discussed below, these estimates will be adjusted down by 25 percent to compensate for double counting of young drug abusers and young problem drinkers.

D. Discussion

Both alcohol abuse and marijuana abuse have significant negative impacts on household income and productivity. Sociodemographic factors which themselves have a strong relationship to labor market success and productivity were carefully controlled in the analyses presented here. The estimated impacts of alcohol abuse and drug abuse are considered to be marginal; that is, they are correlated with the symptoms of alcohol abuse and drug abuse even when other factors hypothesized to be related to alcohol and drug abuse are held constant.

It would be too simplistic to suggest that the impact reflected in the above regression analysis could only be due to alcohol abuse or to drug abuse. Plausible alternative explanations can be offered. One alternative may be that alcohol abuse and drug abuse (either or both) may be symptomatic of other personal problems such as stress or depression. Alcohol and drug abusers may be self-destructive or have other personality disorders, low orientation toward achievement or low motivation. Attitude alone may limit an individual's success in the labor market, regardless of any complications or problems experienced with substance abuse. Extensive research has documented that marijuana use, like alcohol use or abuse of other drugs, is predictable based on social, psychological, and behavioral characteristics observed before the onset of use (Jessor, 1979).

The finding that current marijuana use has little impact on income and that past intensive use has a major impact also merits further discussion. To explain this pattern of results we clearly need a complex causal model that is beyond the scope of this report. It may be that early marijuana use directly causes some reduction in motivation, capacity, or level of performance that cannot be compensated by later achievement. Or, the relationship may be a result of other unmeasured factors. Former intensive marijuana users may maintain counter-cultural values and not seek jobs with high income as the primary reward. They may also have personality characteristics that are not compatible with many jobs.

Jessor (1979) concluded that marijuana use was associated with unconventional and more experimental behavior. Users also place a lower value on achievement and have lower expectations of success. Jessor also cited involvement in other types of problem behaviors though these appear to be part of a constellation of other resultant factors. Behavioral patterns could stigmatize problem individuals and reduce their opportunities to be selected for better jobs.

The analysis has revealed that lifetime, as well as current, problem drinking and marijuana abuse are significant predictors of reduced household income. Use of lifetime indicators is somewhat at odds with current practices of focusing on current or most recent behavior and impacts. The prevailing theory in the analysis of labor market success in this report is that although current earnings are the result of current behavior, they are strongly affected by antecedent life events. These would obviously include variables such as years of formal education, quality and type of education, experience in the work force (analogous to age), lapses in labor force participation, tenure in present job and occupation, and current and past health problems that might impair productivity as well as factors like aptitude and motivation. The study by Bartel and Taubman (1979), discussed earlier) demonstrated that health problems ten or even fifteen years in the past can have significant impacts on current earnings, just as education has a lifelong effect.

The results presented here for both marijuana and alcohol abuse are strongly suggestive of long-lasting impacts of substance abuse on labor market success of individuals and their families. Intoxication can impair the ability to function in school, on the job, or in society. Poor school or job performance can limit future advancement, even if an individual stops abusing alcohol and/or drugs. The initial poor performance can have a permanent affect unless extraordinary measures are taken to counteract the problem (such as remedial or extra education or training). These lifetime impacts of ADM were briefly addressed in the 1981 study on ADM costs to society.

Finally, the social costs of both alcohol and drug abuse have been estimated separately. However, as many recent studies have shown, individuals with problems with alcohol or marijuana also tend to use other substances. For example, in a study of adolescents in 1978 (Rachal et al., 1980), about half of the heavier drinkers also used marijuana at least once a week. Of the youth who used marijuana more than once a month, a third also were classed as heavier drinkers. Of those who reported they had alcohol-related problems, half also reported current marijuana use. These data indicate that there is considerable overlap in the kinds of problems that can affect productivity.

Half or more of young problem drinkers may also have a marijuana problem. Similarly, half of the young marijuana abusers are likely to be alcohol abusers. Thus, the cost estimates for alcohol and drug abuse count many people twice. Assuming this is correct, we reduced each estimate for the age groups between 18 and 34 by 25 percent.

Unfortunately, national surveys of alcohol abuse (Clark and Midanik, 1982) and drug abuse (Miller et al., 1983) included adequate measures for either alcohol or marijuana use, but not both. In order to better gauge the independent and combined effects of alcohol and marijuana, a study must include measures of use and associated problems for both substances.

Referring back to table A-4, for an example, note that 21 percent of males aged 20-24 were estimated to be problem drinkers, and 21.8 percent were estimated to be problem marijuana users. With no overlap, 42.8 percent of this group would be in one or the other of our problem groups. However, with half of each group also in the other group, 10.5 percent are only problem drinkers, 10.7 percent are problem drinkers-users of marijuana, and 10.9 percent are only problem drug abusers, for a total impairment rate in the age/sex group of 32.1 percent, or only three quarters of the duplicated total.

It remains a challenge to future researchers to discover the nature of the relationships among alcohol and drug abuse, attitudes, values, personality, basic socioeconomic and sociodemographic characteristics, and success in the labor market.

SECTION B: FETAL ALCOHOL SYNDROME

The risk of alcohol-related birth defects is causing increasing concern in the field of substance abuse. Although only identified in the early 1970s, research over the last 10 years has conclusively established the fetal alcohol syndrome (FAS) as one of the most serious sets of birth defects and one with a relatively high incidence rate for its degree of severity.

A. Incidence of FAS

Varying estimates on the incidence rate of fetal alcohol syndrome exist. Clarren and Smith (1978) estimate the number of children with FAS in the United States at between 1 and 2 live births per 1,000, with partial expressions at between 3 to 5 live births per 1,000. In a Seattle sample, Hanson, Streissguth, and Smith (1978) reported a rate of 1 in 750 live births. A study in Sweden detected 1 FAS infant per 600 live births (Olegard et al., 1979), and in Northern France, FAS was exhibited in 1 per 1,000 births (Dehaene et al., 1977).

1. Numerous studies have explored the relationship between alcohol consumption and birth defects.
2. Studies have also indicated that risks to the developing fetus are further complicated by mothers' smoking in addition to consuming alcohol (Landesman-Dwyer and Emanuel, 1979).
3. Consistent findings of decreased birth weight, length of torso, head circumference, significantly lower I.Q.'s, cardiac effects, etc. have been documented. Table B-1 indicates the most consistent features of FAS (Clarren and Smith, 1978; Jones and Smith, 1975; Kaminski et al., 1978; Loser and Majewski, 1977).

The risks of stillbirth and spontaneous abortion have also been studied, although limited data are available. Kaminski et al. (1978) has reported that the risks of stillbirth are increased more than 2½ times for women who reported drinking an average of 3 or more drinks per day, smoked, came from lower socio-economic classes, had more prior pregnancies, or were older. A California study detected a significant increase in spontaneous abortions which increased with the amount of reported maternal drinking even after adjusting for variables correlated with drinking (Harlap, Shinono, and Ramcharan, 1979). In a study of 3 New York hospitals, Kline et al. (1980)

Table B-1

Most Consistent Features of the Fetal Alcohol Syndrome

Growth and Performance

- Prenatal onset growth deficiency, more pronounced in length than in weight
- Concomitant microcephaly (small head circumference) even when corrected for small body weight and length
- Postnatal growth deficiency in weight and length, usually below 3rd percentile
- Delay of intellectual development and/or mental deficiency (mean IQ from Seattle study = 64, range 16-92)
- Fine motor dysfunction (poor coordination)

Head and Face

- Microcephaly
- Short palpebral fissures (narrow eye slits)
- Midfacial (maxillary) hypoplasia (underdevelopment of midfacial region)
- Flattened, elongated philtrum (middle of upper lip) associated with thin, narrow vermilion lip borders (highly specific to FAS)
- Minor ear anomalies including low set ears

Limbs

- Abnormal creases in the palm of the hand
- Minor joint anomalies
    - syndactyly (fingers or toes joined together)
    - clinodactyly (abnormal bending of fingers or toes)
    - camptodactyly (one or more fingers constantly flexed at one or more phalangeal joints)

Heart

- Ventricular and atrial septal defect (valve defects)

Brain

- Absence of corpus callosum
- Hydrocephalus (excess fluid in cranium)
- Brain cell migratory abnormalities

Other

- Minor genital anomalies
- Hemangiomas (benign tumors made up of blood vessels) in infancy

SOURCE: Data from Kenneth L. Jones and David W. Smith, The fetal alcohol syndrome. *Teratology*, 12(1):1-10, 1975.

reported five times the risk of spontaneous abortions when 1 ounce of absolute alcohol was consumed twice a week. Harlap et al. also reported a significant increase for second trimester abortions when 1 to 2 drinks were consumed per day. Both of these studies had controlled for confounding variables such as smoking.

Several studies have reported increased risks of physical malformations while indicating that FAS children with the most severe physical signs typically show the greatest degree of mental impairment (Hanson et al., 1978; Majewski et al., 1976). Although few FAS children have been studied as they grew older, Seidenberg and Majewski (1978) have reported that most continue to function at the same mental level even when good foster care and special school programs are provided.

Overall estimates for the risks of drinking during pregnancy are also limited. Oulette et al. (1977) has reported a 37 percent rate of major and minor birth defects for heavy drinkers as compared to 14 percent for moderate drinkers and 9 percent for light drinkers.

Recent discussions with staff of NIAAA and physicians researching FAS suggest that a 1 in 1,000 rate of incidence is a conservative figure. In fact, it has been suggested that a mid-range estimate for the incidence rate is 1 in 500 to 600 births, and that when children having only some, but not all, of the FAS symptoms are included, the upper limit on the incidence rate may be one-half of 1 percent. This 1 in 200 rate is extremely high, and it should be noted that no researchers have suggested that the full fetal alcohol syndrome appears at this rate.

B. Economic Implications of FAS

Marsha Russell (1980) of the New York State Division of Alcoholism and Alcohol Abuse has analyzed the impact of alcohol-related birth defects on New York State. Russell's article carefully reviewed the state of knowledge about alcohol-related birth defects and identified limitations in knowledge about fetal alcohol syndrome. On the basis of that work, a more recent review of the literature on FAS and contacts with leading researchers, this study offers order of magnitude estimates of the national cost of FAS, although a precise incidence rate has not yet been proposed.

Specific birth defects that occur as part of the fetal alcohol syndrome have been identified, along with the type of health treatment that would be required in order to meet the needs of the FAS child, the length of the treatment period, and the age at which the treatments are likely to be necessary. Estimated costs of particular treatments are also presented. These values are presented in table B-2. The specific defects have been numbered from 1 through 10, and treatment regimens are summarized in columns B, C, and D.

Since elements of the treatment and the age at which they would occur are specified along with the treatment costs and the probability of occurrence in a specific FAS case, it is possible to construct the cycle of treatments that a person with FAS would receive at different periods in his/her life. It is also possible to prepare an expected value of treatment received during specific age periods. This was calculated by identifying the treatments that a person would receive at each age and multiplying the cost of these treatments by the probability of occurrence.

It is expected that 80 percent of FAS children have prenatal and postnatal growth retardation that requires neonatal intensive care and a hospital evaluation (Russell, 1980). All infants should also be screened for potential audiological defects while in the intensive care unit (personal communication, Michael Church).

Auditory problems have been associated with fetal alcohol syndrome. Studies have shown that 56 percent of FAS diagnosed children will require a series of audiological evaluations in the first year of life with annual checkups through adolescence, and corrective surgery. Thirty-three percent of FAS diagnosed children may also need hearing aids to correct for mild hearing loss (Gerkin et al., 1984) (personal communication, Michael Church).

FAS children are expected to have some degree of mental impairment, ranging from minimal brain dysfunction (MBD) to severe/profound mental retardation. Fifty-two and one-half percent of FAS children are expected to have learning difficulties related to deficiencies in attention, concentration, and memory, plus a history of hyperactivity and impulsivity. Problems in attention, behavior, and learning have been designated as minimal brain dysfunction (Russell, 1980; personal communication). It is expected that MBD children would require some form of remedial education such as special education in a "resource room" environment in the public schools at an annual cost of $7,400 (Kahalik et al., 1981). It is also expected that these children would require services throughout their years of school attendance (personal communication, Anne Hocutt of the Research Triangle Institute).

Table B-2

Lifetime Cost Estimates of Specific Birth Defects Associated with the Fetal Alcohol Syndrome

(A) Specific Birth Defect		(B) Annual Cost of Treatment	(C) Length of Treatment Period	(D) Lifetime Cost	Estimation of Prevalence in FAS (percent)
1. Prenatal growth retardation		Neonatal intensive care: 7 days at $500 per day[a]	Once	$ 3,500	80
2. Postnatal growth retardation		Hospital evaluation $750	Once	750	80
3. Audiological deficits[b]	A.	Neonatal intensive care: auditory screening at $105/procedure	Once	105	100
	B.	Series of audiological evaluations: 1st evaluation at $45/visit, subsequent evaluations at $25/visit			
			Every 3 months for 1st year of life	120	56
			Semi-annually through adolescence	600	56
Serious otitis media	C.	Surgery (myringotomies) at $400/physicians fee[c]	Once	400	56
Mild sensori-neural hearing loss	D.	Hearing aids at $500/aid including office visits	Through ages 10-12	500	33

(continued)

(A) Specific Birth Defect	(B) Annual Cost of Treatment	(C) Length of Treatment Period	(D) Lifetime Cost	Estimation of Prevalence in FAS (percent)
Mental impairment:				
4. Minimal brain dysfunction	Special education: "Resource room" at $7,400/year[d]	Ages 5-18	103,600	52.5
5. Mild-moderate mental retardation	A. Ambulatory care with special education at $15,000/year	Ages 3 to 21	270,000	45
	B. Home care with day services at $6,200/year[e]	Ages 22-65	272,800	36
	C. Residential care with day services at $18,500/year[f]	Ages 22-65	814,000	9
6. Severe mental retardation	Institutionalization at $25,000/year[g]	Ages 5-65	1,500,000	2.5
7. Heart defects requiring surgery	$17,500 + $750 annual checkup	5 years	21,250	5[h]
8. Cleft palate	Series of operations plus speech therapy	10-15 years	45,000	12.5
9. Kidney defects	Treatment required not yet known		N.A.	N.A.
10. Neurotube	$35,000 per year	5-10 years	$ 262,500	0.5

[a] This is a very conservative estimate; the average length of stay at Columbia Presbyterian Hospital in New York is 19 days (Cheryl Rice, personal communication).

[b] Source: Gerkin, K.P., Church, M.W., and Murrans, L.E., 1984; (personal communication, Michael Church).

(continued)

[c] Cost estimate does not include hospital costs (e.g., per diem rates, operating room charges.)

[d] Source: Kahalik, J.S., Furry, W.S., Thomas, M.A. and Carney, M.F., 1981; (personal communication, Anne Hocutt).

[e] The cost estimate is for day services only. It is based on an average daily rate of $17 per day per client for day services. Individual estimates are not currently available for costs of home care; (personal communication, Charles Lakin).

[f] The type of services provided vary widely. Cost estimates are based on an average daily rate of $51.05 for residential care and day services. Source: 1977 and 1982 National Survey of Residential Facilities for Mentally Retarded People; (Charles Lakin, personal communication).

[g] Actual costs for institutionalization have been estimated as high as $40,000 per individual per year with an average cost of $30,000 in 1984. (North Carolina Department of Human Resources, Division of Mental Health, Mental Health and Substance Abuse Services, personal communication).

[h] This estimate has been revised from a 10% estimate reported by Marcia Russell. Although 10% of heart problems may be associated with FAS, it has been suggested that a more conservative estimate (5%) be utilized to reflect cases actually requiring surgery (Dr. Sterling Clarren, personal communication).

Source: Research Triangle Institute; Russell, 1980.

Forty-five percent of FAS children are expected to have mild to moderate mental retardation which would require provision of ambulatory and residential care with special education between the ages of 3 and 21 years. This has an expected value of $6,750 per FAS child per year (Russell, 1980).

The type of services provided to mentally retarded adults varies widely. Studies have shown that nearly 80 percent of mild to moderately retarded individuals live in the home. Unfortunately, costs of home care are not currently available. However, these individuals would require day services outside the home. These services may include sheltered workshops, adult developmental activity programs, vocational rehabilitation training programs and others with an average daily rate of $17 per client (personal communication, the North Carolina Division of Mental Health and Charles Lakin).

Persons not in the home may receive a wide range of services from several sources. Residential services may include state public residential facilities, group homes, nursing homes, foster homes, "Board and Room," semi-independent accomodations, as well as personal care with an average daily cost of $51.05 for residential care and day services (Bruininks, 1982) (Charles Lakin, personal communication). For the severely/profoundly mentally retarded (2.5 percent of FAS children), lifetime institutionalization may be necessary at an annual cost of $25,000 per FAS child (Russell, 1980).

Neurotube defects and cleft palate have also been associated with fetal alcohol syndrome. Treatment for these defects would begin in the first year of life. One of eight FAS children are expected to have a cleft palate which would require a series of corrective operations and speech therapy with costs estimated at $45,000 over a 15-year period. One-half of one percent of FAS children are expected to have neurotube defects that would require five to ten years of treatment at $35,000 per year. Treatment costs for heart defects are also extensive, with 5 percent of FAS children expected to need corrective surgery and annual examinations for a five-year period (Russell, 1980) (personal communication, Dr. Sterling Clarren).

The expected cost of treatment at a particular age is computed as the probability of each birth defect requiring treatment multiplied by the cost of the treatment. The resulting products are then summed across all defects and treatments. The expected cost of treatment peaks at age 5 at $12,713 per year. Costs remain elevated while children are of school age and begin declining at age 18. Beyond this point, costs are composed of treatment for the mentally retarded: lifetime institutionalization for the severely mentally retarded, and residential care and day services for the mild to moderately retarded.

With the values just described, it is possible to establish some order of magnitude of current costs of FAS in the United States. Again, because there is great uncertainty about the incidence of FAS, results are presented for 3 rates: 1 in 1,000, 1 in 600, and 1 in 200. The 1 in 200 value is much higher than has been suggested by anyone for the incidence of the full fetal alcohol syndrome. Recent findings indicate that an incidence of 1 in 1,000 is probably conservative at this point in time. A mid-range incidence rate for FAS is 1 in 600.

It is assumed in this analysis that the 3 incidence rates selected can be applied to all birth cohorts in the United States. Although this is a strong assumption, it is a useful hypothesis for this exploratory analysis. Future studies must establish the true prevalence of FAS in age cohorts.

Results of the analysis are presented in table B-3. The table reflects the total direct costs of treatment for services delivered whether from the health sector or other sectors, e.g., day services and special education. Assuming a 1 in 1,000 incidence rate, there were approximately 3,600 neonates with FAS in 1980, accounting for health treatment costs of $14.8 million. Across all cohorts, there were 68,000 FAS children aged 18 or under with total treatment costs of $670 million in 1980 ($75 million in health care costs ~~and $590~~ million in educational services), and 160,000 FAS adults with treatment costs of $760 million ($416 million in health services and $344 million in nonmedical services). The 1 in 600 incidence rate implies that there were 380,000 FAS individuals in 1980 with direct services costs of $2.4 billion: 114,000 FAS children with health treatment costs of $125 million, and educational service costs of $990 million; and 267,000 FAS adults with health treatment costs of $574 million, and other service costs of $694 million. At a rate of 1 in 200, there were 340,000 FAS victims between the ages of 0 and 18, requiring $376 million in medical services and $3 billion in educational services. FAS adults numbered nearly 300,000

Table B-3

Expected Cost of Health Treatment in 1980 Due to Fetal Alcohol Syndrome Using Regimen Proposed in Table B-2
($ in millions)

Age	Persons (thousands)	Annual Expected Cost of Treatment (dollars)	Likely Treatments at Age	Total Cost at Prevalence Rate: Conservative 1/1,000 Persons	Conservative 1/1,000 Cost	Mid-Range 1/600 Persons	Mid-Range 1/600 Cost	High 1/200 Persons	High 1/200 Cost
0	3,598	$ 4,122	1,2,3A,3B,8,10	3,598	$ 14.8	6,009	$ 24.8	17,990	$ 74.2
1	3,212	578	3B,8,10	3,212	1.9	5,364	3.1	16,060	9.3
2	3,212	967	3B,3C,3D,8,10	3,212	3.2	5,364	5.2	16,060	15.5
3-4	6,425	7,328	3B,5A,8,10	6,425	47.1	10,730	78.6	32,125	235.4
5	3,453	12,713	3B,4,5A,6,7,8,10	3,453	43.9	5,767	73.3	17,265	219.5
6-7	6,906	11,876	3B,4,5A,6,7,8,10	6,906	82.0	11,533	137.0	34,530	410.1
8-9	6,906	11,701	3B,4,5A,6,7,8	6,906	80.8	11,533	135.0	34,530	404.0
10-14	17,846	11,663	3B,4,5A,6,8	17,846	208.1	29,803	347.6	89,230	1,040.7
15-18	16,483	11,260	4,5A,6	16,483	185.6	27,527	310.0	82,415	928.0
19-21	13,143	7,375	5A,6	13,143	96.9	21,949	161.9	65,715	484.7
22-64	120,765	4,522	5B,5C,6	120,765	546.1	201,678	912.0	603,825	2,730.5
65+	25,708	4,522	5B,5C,6	25,708	116.3	42,932	194.1	128,540	581.3
TOTAL	227,657	$404,638	-	227,657	$1,426.6	380,187	$2,382.4	1,138,285	$7,133.1

Source: Treatment regimen and costs proposed in table B-2; alternative prevalence rates suggested in discussions with Dr. Robert Sokol; computations by Research Triangle Institute; U.S. DOC, U.S. Bureau of the Census: Statistical Abstract of the United States, 1982-1983.

with health care costs of $1.7 billion and nonmedical services of $2 billion. Costs are quite significant by these calculations.

The potential indirect costs from fetal alcohol syndrome are not quite as large as our estimates of the direct health treatment expenditures. The mental retardation associated with FAS means that these individuals are unlikely to achieve a level of productivity comparable to the general population. The 2.5 percent of FAS victims that are severely mentally retarded and, consequently, institutionalized, will certainly never be full participants in the work force. The 45 percent that experience mild to moderate mental retardation not requiring institutionalization will, nonetheless, be much more limited than others in their age cohorts. They are likely to be partially disabled and may, at best, be able to function in a sheltered work environment. The 52.5 percent of FAS victims characterized as having MBD are also expected to have reduced levels of productivity.

Each level of mental impairment has been assigned an expected level of productivity reduction. The severe to profoundly mentally retarded are expected to be completely disabled (i.e., with a 100 percent reduction in productivity). The moderately retarded are considered to be 50 percent impaired, and the mildly retarded are assigned an impairment of 25 percent. (These rates are based on government classifications of moderately retarded individuals as "trainable" and the mildly retarded as "educable;" North Carolina Division of Mental Health, personal communication.) A productivity reduction of 10 percent is assumed for those with minimal brain dysfunction.

The worst case scenario for productivity losses by FAS victims occurs when we apply the high incidence rate of 1 in 200 births to the population aged 18 to 64. This computation suggests that 671,000 adults would have impaired productivity in 1980. Using appropriate values for market and household productivity, and labor force participation, the indirect costs would be $2.6 billion. In comparison, this study has estimated other current productivity losses (excluding mortality) due to alcohol abuse at $55 billion, those due to drug abuse at $30 billion, and those due to other mental illnesses at just over $20 billion.

Applying the 1 in 1,000 incidence rate to the adult population provides an alternate estimate of the indirect costs of fetal alcohol syndrome. This rate suggests that there would be 133,000 FAS adults. Using the same impairment rates, the indirect productivity losses would be $510.5 million.

The mid-range losses are estimated at $853 million, with 225,000 FAS victims of working age (18 to 64 years).

Of particular relevance in the estimation of lost productivity due to mental impairment is the proportion of mental retardation in the general population that is actually attributable to fetal alcohol syndrome. A 3 percent incidence rate is often cited as a reasonable estimate of the proportion of mentally retarded individuals in the adult population of the United States (N.C. Division of Mental Health, personal communication). However, some researchers have suggested that this figure represents an overestimate and that a 1 or 2 percent rate is more representative. On the basis of previously cited information that approximately 360,000 individuals or 20 percent of the mentally retarded are in residential care, a total estimate of 1.8 billion mentally retarded individuals in the United States can be calculated. U.S. Census Department figures estimate that there are 163 million adults over the age of 18 in the United States, allowing the calculation of a 1.1 percent incidence rate of mental retardation in the adult population (U.S. DOC, BOC, 1982). Using a mid-range estimate of 1 in 600, table 8-4 illustrates that there are 107,000 mentally retarded adults (profound/severe, moderate or mild) whose mental impairment is directly attributable to fetal alcohol syndrome. This represents 5.6 percent of all mental retardation in the United States (utilizing the conservative estimate of 1.8 million mentally retarded adults). Alternatively, a 3 percent prevalence rate of mental retardation due to FAS can be calculated using the 3 percent (4.9 million adults) prevalence rate of mental retardation in the adult population.

At the present time, it does not seem possible to create better estimates of the indirect productivity losses due to fetal alcohol syndrome. It is believed that use of the high FAS incidence rate of 1 in 200 births establishes an absolute upper bound on these costs. Without information on the prevalence of FAS in adult cohorts and the degree of impairment actually experienced by these individuals, it is extremely difficult to determine how much lower these costs might be. The 1 in 600 FAS incidence rate is a current estimate. On the basis of the 1 in 600 rate, the fetal alcohol syndrome appears with Down's syndrome and spina bifida as a leading cause of birth defects.

The range of cost estimates presented above has been developed in order to illustrate the costs to society due to fetal alcohol syndrome.

Table B-4

Lost Employment and Reduced Productivity in 1980: Adult FAS Victims, Number of Persons, and Value of Lost Employment
(Number in thousands, $ in millions)

Mental Impairment	Total Lost Productivity at Prevalence Rate					
	Conservative 1/1000		Mid-Range 1/600		High 1/200	
	Persons	Value	Persons	Value	Persons	Value
Minimal brain disfunction	70	$130.3	118	$217.7	352	$ 651.5
Mild mental retardation	52	240.7	87	402.2	260	1,203.5
Moderate mental retardation	8	77.2	14	129.1	42	386.3
Severe/profound mental retardation	3	62.3	6	104.3	17	312.1
All FAS	133	510.5	225	853.3	671	2,553.4

Source: Research Triangle Institute.

The model has been constructed in such a way that it can be readily modified to improve the precision of estimates when more and better information on the incidence of FAS in specific birth cohorts, the prevalence of particular birth cohorts within the fetal alcohol syndrome, the treatment regimen appropriate for these defects, and the cost of these treatments is available.

C. RESEARCH DEVELOPMENTS ON THE RELATIONSHIPS AMONG MENTAL ILLNESS, DRUG ABUSE, AND CRIME

SECTION C: RESEARCH DEVELOPMENTS ON CRIME AND MENTAL ILLNESS AND DRUG ABUSE

A. Introduction

This section offers some new research findings about crime and mental illness and drug abuse. The new contributions have led to improved estimates of the economic impact of these disorders on society. The findings were incorporated in the cost estimation of chapter III. This discussion is intended to offer further insights to the rationale behind the cost estimation.

B. Mental Illness and Crime

The approach to estimating costs of crime associated with mental illness is outlined in two sections below. In section one, the rationale and approach for estimating criminal justice system costs associated with mental illness and public order crime are presented. In section two, the reasons for not attributing costs associated with violent and property crime to mental illness are discussed.

1. Mental Illness and Public Order Offenses

Two recent developments have changed the way we deal with the mentally ill in U.S. society. Deinstitutionalization and stringent civil rights statutes limiting the involuntary commitment of those thought to be mentally ill have meant fewer individuals are hospitalized in mental health facilities (McGarry et al. 1981; Roth, 1980; Shah, 1981). One result of deinstitutionalization and limited civil commitment has been that more individuals who exhibit mental disorder symptoms are dealt with by the criminal justice system.

Eaton (1980) uses the term "bizarre behavior" to refer to human activities that are odd, incongruous, unexpected, or culturally deviant. Bizarre behavior, often taken to be an indication of mental illness, is also likely to attract the attention of the police or private citizens who are concerned, frightened, or disturbed by the behavior and notify the police. Those who act in a disruptive or troublesome manner have always been subject to detainment and arrest. Since deinstitutionalization and tightened requirements for involuntary commitment, more of the mentally ill are in the community and subject to arrest. Adler (1981) discusses this "hospital to jail" phenomenon.

In a study of mentally ill offenders in Lucas County, Ohio, the authors argued that "Potential, past and current mental health patients are often converted to the status of criminal offenders on the basis of minor misconduct" (Ventura and Jacoby, 1983:1). Legislation in Ohio and other states was designed to minimize institutionalization and protect patients' rights had reduced mentally ill inpatient populations. Then, however, voluntary and involuntary commitments often could not be made because the number of treatment slots had been reduced and because the potential patient could not be shown to be a danger to himself or others. Ventura and Jacoby proposed that the police be allowed to hold the apparently mentally ill in the local jail to gain time to find a suitable alternative or until a crisis has passed.

There is considerable variation across the United States in legislation and policies toward institutional treatment and involuntary commitment of the mentally ill. The scenario described above for Lucas County, Ohio may not be exactly replicated in other jurisdictions. It seems clear, however, that, in the face of mental hospital deinstitutionalization and limits on involuntary civil commitment, the mentally ill have often come to the attention of the police in a situation requiring intervention by the criminal justice system.

The manner and conditions under which the deinstitutionalized mentally ill encounter the criminal justice system suggest how the costs might be estimated. The scenarios described above indicate that the mentally ill are most likely to be arrested for offenses of disorderly conduct, vagrancy, and the set of public order offenses recorded in the Uniform Crime Reports as "other offenses." Arrests for those categories totalled 2.575 million in 1980, or 24.7 percent of all arrests. Implications of the citations above are that an appreciable although relatively low proportion of arrests for public order offenses was due to the exhibited behavior of the deinstitutionalized mentally ill persons.

This study makes the conservative assumption that 10 percent of arrests for these public order offenses represents the number of mentally ill who are processed by the criminal justice system.

2. Mental Illness and Violent and Property Offenses

Collins and Schlenger (1983), Hare (1983), and James et al. (1980), using systematic diagnostic methodologies, found high rates of psychiatric disorder among prison inmates. The Collins and Schlenger findings, for example, showed that 77.5 percent of 1,149 male felons entering North Carolina prisons between March and May, 1983 had, in their lifetimes, received at least one psychiatric disorder diagnosis. This rate was twice as high as that for males in the Baltimore, New Haven, and St. Louis communities. In spite of this evidence that the prevalence of mental disorder is higher among criminal offenders than in the general population, no violent or property crime costs are assigned to mental illness.

There are two reasons for not assigning a crime causation factor to mental illness in spite of evidence that offenders are more likely to be diagnosed as having a psychiatric disorder than the general population. The first reason has to do with double counting. Substantial percentages of the psychiatric disorder diagnoses found among offenders are for alcohol abuse/dependence and drug abuse/dependence. Lifetime prevalence disorder rates for these categories were 49.5 percent and 18.8 percent in the North Carolina inmate sample. Alcohol abusers and drug abusers are not distinct from each other or all the mentally ill. Ascribing a criminogenic effect to mental illness would result in double counting because alcohol and drug abuse categories are confounded with other psychiatric disorder categories. Costs of crime associated with alcohol and drug abuse have already been estimated in conjunction with those problems.

The second reason for not ascribing crime costs to mental illness is the absence of any systematic explanation of how mental illness, other than that connected with alcohol or drug abuse, is responsible for involvement in criminal behavior. Schizophrenia, major depressive episodes, and obsessive compulsive disorders are not viewed as criminogenic. One disorder category that is associated with involvement in crime is antisocial personality. This disorder, however, is very often accompanied by an alcohol and/or drug abuse problem (American Psychiatric Association, 1980; Hare, 1983); therefore, the double counting of criminogenic effects is again an issue for this disorder type.

C. Drug Abuse and Violent Crime

In the 1981 report on the economic costs of ADM (Cruze et al., 1981), robbery was the only violent crime associated with drug abuse. Studies performed prior to 1981 indicating a causal connection between drug abuse and nonacquisitive violent crime were not convincing. It was and is clear that daily users of heroin engage in a great deal of property crime to help finance expensive drug habits, but it was not clear that drug abuse explained significant numbers of nonacquisitive violent crimes such as homicide and assault.

Recent evidence suggests that significant numbers of homicides and assaults are caused by drug abuse. The drug abuse-violence connection is seen to occur in two ways: (1) between participants in the illegal drug distribution system to control or expand market share or to retaliate for being "ripped off" in a drug transaction, and (2) as a pharmacologic effect of the abuse of certain single nonnarcotic drugs and polydrug use.

Violence in illegal drug distribution appears to be a serious problem in certain cities. Swersey (1981) examined the increase in homicides in Harlem from 1968 to 1973. He found that the increase was largely a function of an increase in deliberate killings--many of them drug-related. McGuire (1983) reports an analysis of the circumstances of 1,656 homicides that occurred in New York City in 1981. Interviews with investigating police officers resulted in 23.7 percent being classified as drug-related homicides. Zahn and Bencivengo (1974) found that, between 1969 and 1972 in Philadelphia, homicide between drug users increased appreciably and in 1972 represented 30.8 percent of all homicides occurring in the city. Wish et al. (1981) analyzed data on arrestees in Washington, DC and found that, contrary to beliefs that drug users tend not to be involved in assaultive crimes, drug positive arrestees were arrested for homicide and assault at about the same rate as nonusers. Monforte and Spitz (1975) found that 43 percent of 753 homicide victims in Detroit in 1973 were narcotics users and that the percentage of homicides connected with narcotics traffic is very high. Ongoing work by McBride (1983) using data for Miami, Florida shows that drug distribution system violence is a very serious problem.

It is clear from the evidence that drug distribution system violence varies across locations and across time periods. Riedel and Zahn (1983) studied homicide in eight American cities for 1978. They found considerable variation in the percentages of homicide victims found to have narcotics in their systems at medical examiner testing. The 1978 results for Philadelphia, one of the eight cities, showed the presence of narcotics in homicide victims was less than it had been in the earlier Zahn and Bencivengo (1974) study. Swersey's (1981) study of Harlem homicide also indicated a decrease in homicides from 1973 to 1974 after several years of increase. This over-time and over-location variation for drug distribution system violence makes the choice of a single causal estimate difficult.

There is evidence that the pharmacologic action of some drugs or drug combinations, in conjunction with other factors, is related to violent behavior. Although the evidence of an amphetamine relationship to violent behavior is not consistent, Ellinwood (1971) and Asnis and Smith (1978) found a direct relationship between amphetamine use and assaultive behavior. Tinklenberg (1973) found a clear association between barbiturate use and assaultive behavior. Chaiken and Chaiken (1981) also found a barbiturate use-assaultive crime relationship in a sample of prison inmates. Collins (1982) found that in a drug abuse treatment population, those whose primary drug problems were identified as amphetamines or barbiturates were significantly more likely to report assaultive behavior than were those with other types of drug problems.

The 1979 Survey of Inmates of State Correctional Facilities (U.S. DOJ, BJS, 1983c) found that 30 percent of persons incarcerated for violent offenses had been under the influence of some drug at the time of the crime.

The use of multiple psychoactive substances has become very common in recent years (Bray et al., 1982; Fishburne et al., 1980; Johnston et al., 1981). Drug abusers mix different drug types on the same drug use occasions and switch from one drug to another in serial fashion. This polydrug use pattern increases the potential for unpredictable behavior as a result of drug interaction effects. Very little information exists about the behavioral effects of polydrug use, so it is somewhat speculative to attribute violent behavior to the use of multiple drugs. However, in combination with the evidence already cited, the potential for drug interaction effects that produce violent behavior is an additional reason for changing the position in the 1981 report that attributed no drug abuse-violent crime effect.

It is the position of the present report that 10 percent of homicides and serious assaults are caused by drug abuse. The relationship is ascribed to drug distribution system violence and pharmacologic effects of barbiturate, amphetamine, and polydrug use with drug distribution system violence deemed most important. This 10 percent attribution is considered a conservative assumption made in the absence of appropriate data. The studies cited above offer strong evidence about the involvement of drug abuse in violence; however, the extent of the relationship is not well determined.

D. ADMINISTRATIVE COSTS OF PUBLIC SOCIAL WELFARE PROGRAMS

SECTION D: ADMINISTRATIVE COSTS OF PUBLIC SOCIAL WELFARE PROGRAMS

Just as various levels of government assist ADM victims in paying for their health care treatment, public social welfare programs are available to supplement the income of ADM victims that are disabled for some period of time. The major programs that provide assistance are listed in table D-1 and include unemployment insurance, disability payments, veterans pensions and compensation, workers compensation, and supplemental security income, among others. These programs accounted for cumulative expenditures of $65 billion in 1980. This is slightly more than half the contribution of governments to payments for personal health care expenditures.

The purpose of these social welfare programs is to assist individuals who suffer from both long- and short-term problems and have low earnings and/or income. In determining whether needy individuals are eligible for particular programs, information is filed on the nature of problems experienced by individuals. This information makes it possible to identify program expenditures that are due to ADM problems of beneficiaries.

Within these social welfare programs, a distinction must be made between two functions. First of all, the programs transfer income to needy individuals to make up for earnings or income that the individuals cannot provide for themselves. This component of program expenditures constitutes a transfer of income from the government (and taxpayers) to program-eligible individuals. This issue is discussed further in section E of this appendix. Secondly, there are real costs (administrative expenses) in effecting the transfer of income.

Total ADM-related administrative costs for these programs was $241 million in 1980 (Social Security Office, personal communication) (table D-1). Of this value, $201 million was associated with mental illness, $2 million with drug abuse, and $38 million with alcohol abuse. This constitutes approximately 6.6 percent of the total cost to administer public social welfare programs to all recipients. Excluding unemployment insurance and workers compensation, total administrative costs were $1.4 billion. Mental illness accounted for almost 15 percent of all affected program administration costs. Alcohol abuse represented about 2.5 percent of these costs. The impact of drug abuse was negligible.

Table D-1
Public Program Social Welfare Administrative Expenditures and Costs Due to ADM Problems, 1980
($ in millions)

Program	Total Expenditures[a]	Percent Administrative Costs	Total Administrative Costs[a]	Costs Alcohol Abuse		Costs Drug Abuse		Costs Mental Illness	
				Percent	Amount	Percent	Amount	Percent	Amount
OASDHI-disability payments	$15,437	2.4	$ 370	3.4	$12	c	$b	8.9	$ 33
Unemployment insurance	16,503	11.0	1,815	-	-	-	-	-	-
Railroad temporary disability insurance	64	7.8	5	3.4	b	c	b	8.9	b
State temporary disability insurance[d]	1,330	3.2[f]	43	3.4	1	c	b	8.9	4
Workers compensation[d]	9,500	4.4[f]	422	-	-	-	-	-	-
Public assistance[e]	1,219	15.3	186	4.8	9	0.5	1	4.7	9
Supplemental Security Income	7,486	8.8	655	0.8	5	c	b	18.8	123
Food stamps	644	4.4	28	4.8	1	0.5	b	4.7	1
Veteran's pensions and compensation	11,306	0.8	90	9.7	9	0.9	1	23.6	21
Vocational rehabilitation[d]	976	4.7[f]	46	-	-	-	-	19.3	9
Total	$64,513		$3,661		$38		$2		$201

[a] Source: Social Security Administration, Office of Retirement and Survivors Insurance, personal communication.

[b] Less than $.5 million.

[c] Less than .0005.

[d] Excludes hospital and medical benefits.

[e] Excludes vendor medical payments and social services.

[f] Percent does not exclude hospital and medical program administrative costs.

(-) No causal relationship is assumed, or no beneficiaries reported ADM as primary cause of eligibility.

Totals may not add due to rounding.

Source: Research Triangle Institute.

E. OTHER IMPACTS OF ADM ON THE ECONOMY

SECTION E. OTHER IMPACTS OF ADM ON THE ECONOMY

A. Introduction

Consumers spend billions of dollars on alcoholic beverages and illicit drugs; a substantial proportion of property crime is committed by drug addicts; and ADM victims receive benefits from social welfare programs such as health treatment through Medicare. These expenditures might be considered to have impacts on the economy though they are intrinsically different from the costs measured in this report because they are indirect or are transferred resources rather than lost resources. The discussion in the following sections is presented to allow the public and policymakers to more completely assess the role of ADM in the economy although the costs discussed are not included in the framework established in this report to calculate the social cost of ADM.

B. Expenditures on Alcoholic Beverages and Illicit Drugs

Total legal alcoholic beverage production and sales in 1979 were $45.0 billion (U.S. DOC, Bureau of the Census, 1982); approximately 50 percent of the consumption was beer, 39 percent was distilled spirits, and 11 percent was wine (DeLuca, 1981). Illegitimate production of alcohol is believed to be negligible, but illicit consumption of alcohol merits attention. It is illegal for those underage to consume alcoholic beverages, and it is illegal to sell them alcoholic beverages. Among high school students in 1978, 31.2 percent were misusers of alcohol, 51.7 percent drank but did not misuse, and 17.2 percent abstained. Male misusers reported consumption of 3.56 gallons of ethanol annually (1.25 ounces of ethanol, or approximately 2.5 "drinks" per day); female misusers reported consumption of 2.59 gallons annually. Male and female drinkers who did not misuse consumed .74 and .48 gallons annually, respectively (Rachal, Maisto, Guess, and Hubbard, 1982). The U.S. Department of Commerce (1982) reports 13 million 15- to 17-year olds in 1978. These figures indicate total annual consumption of 16.5 million gallons (about 2 billion ounces) of ethanol.

Total U.S. sales in 1978 was 2.73 gallons of ethanol for each of the 174 million persons aged 14 or over. When comparing the self-reported consumption of 10th to 12th grade students (excluding 18-year olds) with reported sales of ethanol in the U.S. (DeLuca, 1981), it appears that youth aged 14 to 17 years consumed 3.5 percent of all ethanol in the United States in 1978.

The value of purchases by underage youth is likely to be a smaller proportion of total expenditures than their share of volume of consumption. The contexts for drinking most often mentioned by youth were teenage parties, "hangouts," at home on special occasions, school activities, and in cars at night - all with peers only except at home. These contexts would involve lower prices than locations more typical of adult consumption such as restaurants and cocktail lounges. Furthermore, youth consume a larger proportion of beer, which is a relatively inexpensive source of ethanol.

Surveys of adult populations (age 18 and above) indicate very different drinking patterns than those of teenagers. Between 1971 and 1976 the average reported consumption (using quantity and frequency measures similar to those used to study youth) of all adults ranged from .40 to .50 ounces of alcohol per day (Noble, 1978). At 128 ounces per gallon, this represents approximately 1.3 gallons per year. The reported consumption accounted for only half of what is manufactured and sold. The same results are found for the reported consumption of adults and youth (age 15 to 17).

Lower reports of consumption than sales have been replicated in all of the national surveys, with the under-report ranging from 40 to 60 percent. Because youth aged 15 to 17 reported consuming 3.5 percent of all alcohol sold, this value was adjusted to nearly 7 percent of all alcohol sold.

Estimates of illicit drug use have been made by two government agencies: an interagency task force entitled the National Narcotics Intelligence Consumers Committee (NNICC); and the Internal Revenue Service. Both estimates used similar estimation methodologies and the same data sources.

NNICC estimated that the retail value of illicit drug consumption in 1979 ranged from $56 to $74 billion (see table E-1). Consumer expenditures for cocaine were $21.7 billion; for marijuana, $18.7 billion; for heroin, $8.5 billion; for other drugs, $16 billion. Volume of consumption was also estimated. The NNICC values represented the value of drugs consumed at "street" prices by retail customers and at "wholesale" prices by the dealers themselves.

The Internal Revenue Service estimated the value of marijuana, cocaine, and heroin only. These values were $8.6 billion, $5.7 billion, and $7.2 billion, respectively (see table E-1). Again, this represents the value of drugs consumed at "street" prices and value of drugs consumed by dealers. These values are somewhat lower than the corresponding NNICC estimates for

Table E-1

Consumption of Illicit Drugs in 1979 as Estimated by NNICC and the IRS

Type of Drug	NNICC Quantity (metric tons)	NNICC Retail Value ($ in millions)	IRS Quantity (metric tons)	IRS Retail Value ($ in millions)
Heroin	3.4-4.0	$7,790-9,160	1.9-4.5	$1,160-13,260
Cocaine	25-31	19,500-24,180	5.2-12.8	3,280-8,080
Marijuana	10,000-13,600	15,480-21,930	6,647	4,490-12,650
Hashish	200	1,480	N.E.	N.E.
Dangerous Drugs	-	12,000-17,000	N.E.	N.E.
Total		$56,250-73,750		$8,930-33,990

N.E.: Not Estimated.

Sources: NNICC (National Narcotics Intelligence Consumers Committee) and Internal Revenue Service, Department of the Treasury, 1983.

marijuana (by 54 percent), cocaine (by 74 percent) and heroin (by 15 percent). Differences between NNICC and the IRS are due primarily to differences in estimating the consumption of marijuana (the IRS is lower by 44 percent), cocaine (by 68 percent), and heroin (by 14 percent). The balance of the differences in total values is due to differences in the calculation of price.

While the differences between the illicit drug consumption estimates of the NNICC and IRS are appreciable, both estimates are enormous. Both indicate the order of magnitude of the true values and establish a framework for improved estimates in the future.

C. Property Crime Transfers Associated with Drug Addiction

Property crime is one of the leading problems attributable to drug abuse and is of concern to society, the criminal justice system, the drug abuse treatment system, and policy makers. This report has estimated that nearly 35 percent of the costs to society of drug abuse are crime related. The value of property stolen due to drug addiction is relevant to understanding the impact on the economy of drug abuse; but because this value is a transfer payment, it is not directly estimated in this report.

Criminal career costs have been included as a cost to society. This component reflects the value of time diverted from legitimate (market and household) activities and used to pursue income through criminal activities including property crime, drug trafficking, and victimless crimes such as gambling and prostitution. Estimates in chapter III indicate that nearly 1.1 million addicts participated in a criminal career with a corresponding loss to society of $8.7 billion of productive effort.

Evidence on property crime and drug abuse has been collected from several sources. The National Crime Survey included a household component that gathered information on personal victimization by violent and property crimes and established an overall estimate of the value of property stolen in 1980. The survey asked detailed questions on the type of victimization, the time and place of occurrence, the injury suffered, loss of valuables, damage to property, and whether the offense(s) was reported to the police. A series of reports has assessed the extent of personal victimization in the United States on the basis of these data.

The nationally representative survey provides comprehensive estimates of the number and value of crimes for the type of offenses often associated with drug abusers and addicts. Data on robbery, personal larceny, burglary, household larceny, and motor vehicle theft are also available.

There were 32.8 million victimizations in 1980 (see table E-2). The most frequent type of crime was personal larceny, with 14 million occurrences, one per every 16 persons in the U.S. The average value of property and cash stolen was $116 with a total value of $1.6 billion. Burglary resulted in the largest total transfer of $3.2 billion from 6.5 million victimizations. The 1.3 million motor vehicle thefts in 1980 had the largest average loss at $938 per incident. The total value of theft from all types of personal offenses was $7.3 billion. These BJS estimates represent the most comprehensive data to date on the extent and value of personal crime.

The 1981 RTI study reviewed evidence on the drug abuse-property crime link and concluded that one of five property crimes was attributable to drug addiction. Studies of arrestees, prison populations, drug abusers, and treatment populations were reviewed. Attributions to drug abuse were made for 26.8 percent of robberies, 22.4 percent of burglaries and 18.6 percent of larcenies and motor vehicle thefts (see table E-2). Application of these rates to the nationwide value of personal property crime in 1980 yields an estimate of $1.5 billion attributable to drug addiction. This estimate is partial. It does not measure the dollars lost by crime victims to drug abusers, does not include offenses like forgery, fraud and other types of crime, and does not represent income from the illegal drug distribution system or from victimless crimes such as prostitution. These estimates are relatively consistent with other recent research findings on the economic behavior of heroin addicts.

Johnson and Goldstein (1984) estimated that daily heroin users spend $13,189 on the drug per year, with those using heroin three to five days a week spending $6,431 per year on the drug. With an estimate of $10,000 a year as the average annual cost of heroin for addicts, 500,000 addicts spend $5 billion a year for the drug.

Recent findings from the Treatment Outcome Prospective Study (TOPS) indicate that daily heroin users have $8,426 more in illegal income per year than nonusers or nondaily users of heroin (Collins, Hubbard, and Rachal, 1984). Based on an assumption that two-thirds of heroin users are daily users (500,000 X .667 = 333,500), this group has a total of $2.8 bil-

Table E-2

Value of Property Transferred by Personal Crime in 1980; Total and Proportion Attributable to Drug Abuse

Type	Number of Crimes (thousand)	Average Value	Total Value ($ in millions)	Attributed to Drug Abuse	
				Percent	Value ($ in millions)
Robbery	1,138	$ 138	$ 157	26.8	$ 42
Personal Larceny	14,023	116	1,621	18.6	302
Burglary	6,522	490	3,193	22.4	715
Household Larceny	9,787	116	1,138	18.6	212
Motor Vehicle Theft	1,290	938	1,210	18.6	225
Total	32,760	N.A.	$7,319	N.A.	$1,496

Source: U.S. DOJ, BJS, unpublished data, and U.S. DOJ, BJS, NCJISS, 1983 and causal factors from Cruze et al., 1981.

lion in illegal income. For many heroin addicts, this illegal income comes primarily from victimless crime and does not involve the direct victimization of others.

In comparison to extrapolations from Johnson and Goldstein (1984) and Collins et al. (1984), the $1.5 billion loss suffered by victims shown in table E-2 seems small. It should be remembered, however, that the $1.5 billion estimate is for a limited number of offenses and does not include illegal income earned from drug distribution, often a major income source for drug addicts.

D. Public Transfer Payments to ADM Victims

Another impact of ADM on the economy is the social welfare program transfer payments received by ADM victims. Victims are eligible to receive benefits under some social welfare programs. These values are not costs to society using the conceptual framework developed by the Public Health Service (Hodgson and Meiners, 1979). Clients usually qualify for transfer payments because of a consequence of ADM which is considered and totalled as a cost in this report. Adding the value of transfer payments to the cost estimates would result in "double counting" of the problem.

For example. OASDHI-disability payments are made to compensate persons who are unable to work for their loss in earnings. The "lost employment" estimates included in the PHS framework represents the value of productivity that is lost because persons cannot function in the workplace and at home. The value of these transfers can be estimated using information on administrative expenses in public social welfare programs (see section D of this appendix).

Social welfare program transfers excluding unemployment insurance and workers compensation totaled $37 billion in 1980. ADM disorders accounted for $7.5 billion, or 20 percent (table E-3). Mental illness represented $5.6 billion in transfers, just over 15 percent; alcohol represented 5 percent; the impact of drug abuse was negligible.

The administrative costs and the value of transfers from these programs were distributed among alcohol abuse, drug abuse, and mental illness based on varying factors. For programs involving disability payments, the distributional factors were equal to the proportion of patient days attributable to the disorder spent in general hospitals and VA hospitals. More specific data were available for programs related to vocational rehabilitation (U.S.

Table E-3

Public Program Social Welfare Transfer Payment Expenditures and Costs Due to ADM Problems, 1980
($ in millions)

Program	Total Expenditures[a]	Percent Transfer Costs	Total Transfer Costs[a]	Costs Alcohol Abuse Percent	Costs Alcohol Abuse Amount	Costs Drug Abuse Percent	Costs Drug Abuse Amount	Costs Mental Illness Percent	Costs Mental Illness Amount
OASDHI-disability payments	$15,437	97.6	$15,067	3.4	$ 507	c	$ 2	8.9	$1,341
Unemployment insurance	16,503	89.0	14,688	-	-	-	-	-	-
Railroad temporary disability insurance	64	92.2	59	3.4	2	c	b	8.9	5
State temporary disability insurance[d]	1,330	96.8[f]	1,287	3.4	43	c	b	8.9	115
Workers compensation[d]	9,588	95.6[f]	9,167	-	-	-	-	-	-
Public assistance[e]	1,219	84.7	1,032	4.8	49	0.5	5	4.7	49
Supplemental security income	7,446	91.2	6,791	0.8	53	c	2	18.8	1,279
Food stamps	644	95.6	616	4.8	29	0.5	3	4.7	29
Veteran's pensions and compensation	11,306	99.2[f]	11,216	9.7	1,088	0.9	102	23.6	2,651
Vocational rehabilitation[d]	976	95.3	930	-	-	-	-	19.3	179
Total	$64,513		$60,851		$1,771		$115		$5,648

[a] Source: Social Security Administration, Office of Retirement and Survivors Insurance, personal communication.

[b] Less than $.5 million.

[c] Less than .0005.

[d] Excludes hospital and medical benefits.

[e] Excludes vendor medical payments and social services.

[f] Percent does not exclude hospital and medical program transfer costs.

(-) No causal relationship is assumed, or no beneficiaries reported ADM as primary cause of eligibility.

Totals may not add due to rounding.

Source: Research Triangle Institute.

DOC, BOC, 1982) and social security income (U.S. DHHS, SSA, personal communication). These proportions are based on information collected on persons likely to receive assistance from these programs.

E. Sources of Payments for Health Care Expenditures

In the main body of the report, values were assigned to the health treatment services received by ADM victims, regardless of the source of payment. The data on source of payment describe how the cost burden is borne.

How much the government pays for the treatment of ADM victims is a relevant topic for policymaking. The size of the ADM treatment bill will be pertinent to the Federal government and private third-party insurers in deciding whether or not to provide coverage for these disorders.

This question has been addressed in the same fashion as in the 1981 study. The first step was to obtain financial data on health settings specializing in ADM treatment. The NDATUS survey obtains financial information from specialty units which treat alcohol and drug abusers.

The data for alcohol and drug abuse treatment specialty units are presented in table E-4. For units primarily providing alcohol abuse services, the largest single source of payments was private health insurance, at 26 percent of $1.1 billion in 1980. State, Federal, and local governments paid an additional 45 percent, with state government paying 21 percent. Client fees were the next largest source, at approximately 10 percent or $110 million.

Settings offering primarily drug abuse treatment are somewhat different (table E-4). Of funds totaling $534 million in 1980, 7 percent were client fees, 8 percent came from private health insurance, and the remaining 81 percent was from a variety of government sources.

Community mental health centers primarily serve individuals with other mental disorders, although some alcohol and drug abusers are clients. The National Institute of Mental Health has collected information on funding for CMHCs. Their findings are presented in table E-5. Government funds, including grants for various purposes, provided 64 percent of the CMHC resources. Twenty-one percent were Federally funded with 34 percent from the state, and 9 percent from other governmental units. Direct services provided by CMHCs generated 32 percent of revenues (3.7 from patient fees and 7.3 percent from private insurance). The remaining 21 percent was from sources such as Medicare, Medicaid, schools, and other grants.

Table E-4

Funding for All Alcoholism and Drug Abuse Treatment Units
by Source of Funds in 1980
($ in thousands)

Funding Source	Alcohol Abuse		Drug Abuse	
	Amount	Percent	Amount	Percent
ADAMHA block grant	$ 50,910	4.5	$ 67,804	12.7
Other ADAMHA program support	12,133	1.1	11,572	2.2
Other Federal funds	112,456	10.0	46,070	8.6
State government	235,751	21.1	165,412	31.0
Local government	108,254	9.6	41,423	7.8
State/local government	45,413	4.0	16,612	3.1
Social services block grant	13,959	1.2	5,174	1.0
Public welfare	18,257	1.6	17,226	3.2
Public health insurance	77,922	6.9	62,229	11.7
Private health insurance	296,419	26.4	43,513	8.2
Private donations	28,754	2.6	17,358	3.3
Client fees	110,272	9.8	35,588	6.7
Other	12,677	1.1	3,651	0.7
Total	$1,123,175	100.0	$533,631	100.0

Totals may not add due to rounding.

Source: U.S. DHHS, NIAAA (1983); U.S. DHHS, NIDA (1983).

Table E-5

Source of Operating Funds
Federally Funded Community Health Centers
in 1980

Source of Funds	Percent
Government funds	64.2
Federal funds	21.2
Staffing grants	15.2
Construction grants	0.8
Children's grants	1.4
Research and training	0.4
Other Federal funds	3.5
State funds	33.9
Local government funds	8.8
Other government funds	0.2
Receipts from direct services	31.8
Patient fees	3.7
Insurance (priv. & voluntary)	7.3
Medicare	2.9
Medicaid	10.9
Schools	1.0
Title XX	3.7
Other receipts from services	2.2
Receipts from indirect services	0.6
Schools	0.2
Other receipts	0.4
Philanthropy	0.5
Other fund raising	0.8
Other receipts	2.2
Total	100.0

Totals may not add due to rounding.

Source: U.S. DHHS, NIMH (1981).

The types of health treatment settings discussed above represent a small proportion of all treatment services used by ADM victims. Data on the other settings, including general hospitals, physicians' offices, nursing homes, and other health providers and sundries, are maintained for all illnesses on an annual basis. This study has distributed the aggregate expenditures of alcohol abuse, drug abuse, and mental illness for each setting estimated above by the percentage distribution of national funding sources. The costs for alcohol abuse exclude fetal alcohol syndrome because costs were not estimated by setting for this disorder. Tables E-6 through E-8 report the expenditure for each setting and its distribution across out-of-pocket and various third party funds including Federal, state and local, and private. These funds are reported according to the distribution of illnesses being treated in that setting.

For alcohol abuse, of the $8 billion spent in settings for which we have data, the two largest payment sources are Federal funds and private third party funds, each contributing approximately 31 percent. This is followed by individuals, and State and local third party funds which together provide approximately 12.5 percent of all resources.

Drug abuse treatment expenditure patterns are similar. Twenty percent of all payments are from out-of-pocket sources and more than 20 percent from state and local governments. Federal funds and private third party funds each contribute slightly more than 30 percent of the total budget. For mental health treatment, the largest payment source was from Federal funds, with approximately 33 percent. Out-of-pocket sources and private third party funds covered approximately 25 percent each, while state and local governments paid under 20 percent of all costs.

It has also been possible to obtain more specific information on payments from Medicare for ADM disorders. These data overlap with values discussed immediately above and represent the proportion of federal payments for Medicare services in general hospitals.

Unpublished 1980 data on inpatient hospital discharges by Medicare eligible individuals indicate that ADM disorders accounted for 3.8 percent of all discharges (table E-9). Of 10.3 million total Medicare discharges, 129,000 or 1.3 percent were related to alcohol abuse as defined by diagnostic codes. Thirty-seven thousand discharges (0.4 percent) were associated with drug abuse, and 214,000 discharges (2.1 percent) were associated with other

Table E-6

Estimates of Funds for Alcohol Abuse by Setting and Source, 1980
($ in millions)

Setting	Expenditures for Alcohol Abuse Treatment	Out of Pocket	Third Party Funds		
			Federal	State & Local	Private
ADM specialty hospitals[a]	-	-	-	-	-
General hospitals	$4,800	$ 437	$1,982	$ 624	1,747
Other specialty facilities					
CMHCs	181	7	38	81	55
Alcohol abuse facilities	385	38	60	118	169
Other facilities[a]	-	-	-	-	-
Other general services					
Nursing homes	167	70	52	43	2
Physician services	726	271	147	45	264
Dentist services	621	271	13	10	130
Other health professionals	171	101	33	13	24
Drug and drug sundries	750	620	31	30	69
Other health services	359	-	185	80	94
Volunteer services	-	-	-	-	
Total	$8,160	$2,012	$2,541	$1,044	$2,554

Totals may not add due to rounding.

[a] Data not available. Expenditures in ADM specialty hospitals and other facilities were over $6 billion.

Source: U.S. DHHS, NIAAA (1983); U.S. DHHS, NIMH (1981); Gibson and Waldo (1981).

Table E-7

Estimates of Funds for Drug Abuse by Setting and Source, 1980
($ in millions)

Setting	Expenditures for Drug Abuse Treatment	Out of Pocket	Third Party Funds		
			Federal	State & Local	Private
ADM specialty hospitals[a]	-	-	-	-	-
General hospitals	$ 527	$248	$218	$ 69	192
Other specialty facilities					
CMHCs	50	2	11	22	15
Drug abuse facilities	295	20	69	114	92
Other facilities[a]	-	-	-	-	-
Other general services					
Physician services	28	10	6	2	11
Dentist services	59	45	1	1	12
Other health professionals	16	10	3	1	2
Drug and drug sundries	71	58	3	3	7
Other health services	34	-	17	8	9
Volunteer services	-	-	-	-	
Total	$1,080	$193	$328	$220	$338

Totals may not add due to rounding.

[a] Data not available. Expenditures in ADM specialty hospitals and other facilities were over $6 billion.

Source: U.S. DHHS, NIDA (1983); U.S. DHHS, NIMH (1981); Gibson and Waldo (1981).

Table E-8

Estimates of Funds for Mental Illness by Setting and Source, 1980
($ in millions)

Setting	Expenditures for Mental Illness Treatment	Out of Pocket	Third Party Funds: Federal	Third Party Funds: State & Local	Third Party Funds: Private
ADM specialty hospitals[a]	-	-	-	-	-
General hospitals	$5,088	$ 463	$2,101	$ 661	1,862
Other specialty facilities					
CMHCs	997	37	211	446	303
Other facilities[a]	-	-	-	-	-
Other general services					
Nursing homes	2,783	1,166	863	718	46
Physician services	870	315	176	54	326
Other health professionals	184	109	36	14	26
Drug and drug sundries	810	670	33	32	74
Other health services	388	-	199	87	102
Volunteer services	-	-	-	-	-
Total	$11,120	$2,760	$3,619	$2,012	$2,729

Totals may not add due to rounding.

[a] Data not available. Expenditures in ADM specialty hospitals and other facilities were over $6 billion.

Source: U.S. DHHS, NIMH (1981); Gibson and Waldo (1981).

mental disorders. Applying these proportions to the $26.3 billion distributed by Medicare to pay for inpatient hospital stays, $332 million were attributable to alcohol abuse, $95 million due to drug abuse, and $552 million due to mental illness.

These estimates suggest that Medicare payments for alcohol abuse accounted for $342 million out of the federal government total of $1,982 million (17 percent). For drug abuse, the Medicare value of $95 million was 44 percent of federal payments for hospital services. Medicare paid $989 million, or 47 percent of the federal share for mental illness of $2.1 billion.

Table E-9

ADM Related Hospital Loss Reimbursed by Medicare, 1980
(Number in thousands, $ in millions)[a]

	Discharges		
	Number	Percent	Payments
Alcohol Abuse	129	1.3	$ 342
Drug Abuse	37	.4	105
Mental Illness	214	2.1	552
Total ADM	381	3.8	999
All Illnesses	10,334	100.0	$26,300

[a] Costs included are based on hospital discharges.

Source: U.S. DHHS, HCFA, personal communication.

F. ALCOHOL ABUSE RELATED GASTROINTESTINAL TRACT CANCERS

SECTION F: ALCOHOL ABUSE RELATED GASTROINTESTINAL TRACT CANCERS

A. Introduction

Controversy exists on whether alcohol abuse is causally related to cancers of the gastrointestinal tract. While there is strong evidence that malignancies of the liver can be caused by alcohol abuse, research findings on other neoplasms are mixed. Costs of liver malignancies are represented in the main body of the report. In lieu of more definitive findings about alcohol abuse and other cancers, these costs were not included. This section outlines the potential magnitude of costs of other cancers of the gastrointestinal tract.

B. Evidence on the Link

Several studies have been performed in the last 20 years which indicate that alcohol abuse is related to cancer of the gastrointestinal tract. These studies have found a significant increase in risk associated with high levels of alcohol abuse, leading to malignancies of the GI tract. In Alcohol and Health III (Noble, 1978), it was estimated that over 50 percent of primary malignant liver cancers were due to alcohol abuse. However, studies have indicated both higher and lower levels of association. A wide variation has been found in the association of other GI tract cancers to alcohol abuse.

A factor significantly aggravating the relationship of alcohol abuse to cancer is consumption of tobacco. Epidemiological studies indicate that consumption of alcohol and tobacco increases the risk of contracting cancer over that of consumption of only one or the other product.

After studying the smoking and drinking habits of 483 people with cancer of the mouth and pharynx and 447 controls, Rothman and Keller (1972) reported that risks accelerated with increased exposure to each factor. They calculated that 76 percent of the disease in males might be eliminated if exposure to both alcohol and tobacco were avoided. Another study reported that women who drink and smoke heavily may develop cancers of the buccal cavity and tongue 15 years earlier than women who abstain from both (JAMA, 1976).

The means by which alcohol acts on body tissues is still unclear. There are theories that specific ingredients in different types of alcohol are carcinogens. Similarly, the effect of tobacco on body tissues must be better understood to disentangle the combined effects of both of these substances.

Malnutrition and anemia have also been associated with increased risk of oral, hypopharyngeal, and esophageal cancers. Nutritional defects, especially of vitamin-B complex and iron, have been associated with heavy alcohol intake (Wynder and Shigomatsu, 1967). Studies have reported that alcohol impairs the absorption of thiamine in the intestines, and that several intestinal enzymes that aid in the movement of nutritional factors into the blood streams are affected by alcohol (Mezey et al., 1970; Mott et al., 1972; Roggen et al., 1972).

In studies of alcohol related cancer risks, authors have emphasized the need to clearly distinguish among the specific forms, amounts, and duration of drinking involved. Increasing knowledge about how cancer is caused and how it is related to alcohol abuse has not yet produced a consensus on which cancers are caused by alcohol and what proportion of new cancers and of mortalities are related to alcohol abuse.

C. Potential Costs

Nonetheless, it is possible to examine the potential implications for the economic cost of alcohol abuse if alternative causal factors between alcohol and GI tract neoplasms are hypothesized.

A list of GI tract neoplasms that have been related to alcohol abuse is presented in table F-1. The number of mortalities by type of cancer, and the estimated total cost in 1980 for each type of cancer are also presented. These neoplasms caused more than 60,000 premature deaths in that year. Table F-2 presents estimates of the direct treatment costs in specific settings for 1980-1982 for the alcohol abuse-associated cancers of the GI tract. Another major cost which is associated with mortality is lost productivity.

The analyses indicate that if 100 percent of the specified cancers could be attributed to alcohol abuse, then the economic cost to society for alcohol abuse would be close to $900 million for direct treatment costs in 1980 and $4.9 billion for lost lifetime productivity. Should the estimates of the cost of treatment and mortality due to alcohol abuse-related gastrointestinal tract cancers be added to the already-calculated estimates of alcohol-related costs, the new total would be noticeably, but not overwhelmingly, larger.

Table F-1

Numbers and Economic Cost of Premature Mortality Due to Malignant Neoplasms Related to Alcohol Abuse, 1980
($ in millions)

Type	Mortalities			
		Discounted Value		
	Number	(100 percent)	(10 percent)	(56 percent)
Tongue, malignant	1,874	$ 145	$ 15	$ 81
Mouth, floor malignant	509	39	4	22
Mouth, other, malignant	1,399	108	11	61
Pharynx, malignant, oro	1,127	87	9	49
Pharynx, malignant, naso	585	45	5	25
Pharynx, malignant, hypo	634	49	5	28
Pharynx, malignant, unspecified	1,501	116	12	65
Esophagus, malignant	7,985	619	61	347
Stomach, malignant	14,372	1,115	111	624
Rectum, malignant	7,435	577	58	323
Liver, malignant, primary	2,395	186	19	104
Liver, bile duct type[b]	455	35	4	20
Pancreas, malignant	19,640	1,523	152	853
Larynx, malignant	3,412	264	26	148
Total	63,323	4,911	491	2,750

Source: Unpublished mortality statistics from the Vital Statistics files provided by NCHS, present discounted values provided by NCHS.

Table F-2

Direct Treatment Costs for Alcohol-Abuse Related Neoplasms
($ in millions)

General Health Facilities	Year 1980	 1981	 1982
Hospital-based			
Non-Federal community hospitals (excluding psychiatric units)	$548	$588	$671
VA general hospitals and other facilities	24	24	26
Other Federal facilities	19	19	21
Other general health facilities and services			
Nursing homes	92	98	109
Private practice physicians	29	35	32
Other health professionals	19	21	23
Drugs and drug sundries	85	87	89
Other health services	44	46	51
Volunteer services	15	16	17
Total	$874	$934	$1,039

Totals may not add due to rounding.

Source: Research Triangle Institute.

The $874 million of treatment costs for the selected GI cancers in 1980 is somewhat more than 10 percent of total health treatment costs due to other alcohol abuse related illnesses. These cancers represent a more significant although, again, not overwhelming impact on total mortality costs. The additional 63,000 mortalities attributable to alcohol abuse would almost double the number of deaths attributable to alcohol abuse. At 100 percent discount, however, there would be a $4.9 billion increase in mortality costs, already estimated at $13.9 billion. This would be a 35 percent increase in the mortality costs at the 6 percent discount rate.

The combined increase in treatment cost and mortality cost with a 100 percent attribution would be $5.8 billion. This is 6.7 percent of the total value already attributed to alcohol abuse.

At the other extreme, should only 10 percent of these GI neoplasms be attributable to alcohol abuse, then treatment cost would have been $87 million higher in 1980, and mortality cost would have been $491 million higher. The combined increase would be .7 percent of the cost estimated in this study.

Another interesting comparison is made by taking the 56 percent causal factor currently assigned to malignant primary liver neoplasms. Should the 56 percent rate be appropriate for other GI tract neoplasms, then health treatment costs would be $490 million higher, and mortality costs would be $2.8 billion higher. The total increase would be just under 4 percent of the current estimate for alcohol abuse.

SECTION G. PROTOCOL FOR UPDATING COST ESTIMATES

A. Introduction

Having current data about the economic costs of ADM is essential. Because the function of these values indicates the relative impact of the disorders on economic well-being, it is necessary to have relatively current estimates. Only by maintaining current estimates will it be possible to compare the costs of alcohol and drug abuse, and mental illness to updated estimates of the costs of major disease categories produced by NCHS, and new estimates produced for narrowly defined disorders, such as particular infectious diseases.

The cost estimates presented in this volume have been constructed using a methodology that makes it possible to compare the ADM cost estimates with those for other disorders. The concepts and methodology are consistent with those recommended by the Public Health Service (Hodgson and Meiners, 1979) and with the tradition of previous cost of illness studies.

The best way to maintain current estimates of the economic burden of alcohol abuse, drug abuse, and mental illness is to completely reestimate the costs for each year, utilizing the same methodology from year to year, and inserting the most current values from appropriate statistical series. Completely reestimating costs has essentially been the task of the present effort. This is both time consuming and relatively expensive if the sole function is to provide more recent estimates than the last comprehensive study.

A less expensive and less time intensive technique for maintaining current cost estimates is to make year-to-year adjustments in the values based on a small number of factors that have known relationships to the cost estimates. This is both possible and reasonable where there are a relatively small number of factors that could affect the cost estimates significantly. A specific cost estimate will have some imprecision because adjustments will be made for only some of the most important change factors.

In this section, some of the factors that contribute to changes in estimates of the economic costs of ADM are identified, and then a small number of adjustment factors are selected and their application for use in maintaining current cost estimates is specified.

Finally, the economic costs to society of ADM in 1981, 1982, and 1983 are estimated using the protocol developed in this study.

B. Sources of Changes in the ADM Cost Estimates Over Time

The 1981 RTI study developed formulas for estimating the costs of ADM. The elements of those formulas delineate the variety of factors that are significant in making cost estimates. A short list of these includes:

- inflation in wages and prices;
- growth in the population at risk;
- sociodemographic distribution;
- change in the prevalence or incidence rate;
- causal factors; and
- social responses to ADM.

Changes in any and all of these elements are incorporated when completely new cost estimates are made.

Inflation is the most obvious and inevitable source of change in economic cost estimates. Market prices in our society continually change, sometimes rapidly (at over 10 percent per year) and sometimes more slowly (potentially down to no change, or even a negative change). Moreover, wages and different prices may change at different rates in the same period of time. Health prices increased more rapidly than prices of other consumer goods during the last 20 years. Wages and salaries have grown at a rate faster than consumer prices because productivity of the workforce has improved. Although a single inflation rate may be calculated for the gross national product, different sections of the economy have different rates of inflation. Any methodology proposed to adjust for inflation should allow for differences in rates.

As the total population grows, the number of people potentially affected by ADM also increases. However, some sections of the population may be at higher risk than others. The growth in the total population may not reflect change in the numbers in those subpopulations at risk. For instance, total population grew 3.2 percent between 1977 and 1980, while the labor force increased by nearly 8 percent. Thus, the potential for losses from reduced productivity grew by more during that period than did total population. Conversely, a population at very high risk of motor vehicle crashes--18 to 20 year olds--grew by only 3.7 percent over the 3-year period.

The sociodemographic characteristics of the population at risk can be another source of variation in cost, although such distributions may not change substantially over short periods of time. For instance, in calculating indirect losses, there are substantial differences between productivity values for older and younger workers, and males and females. Should the age/sex distribution of ADM change, while the total population prevalence rate remains the same, the cost to society would change.

Potentially, the true incidence and/or prevalence rates for ADM disorders could change from one year to the next. Completely apart from population growth, the proportion of the population at risk that experiences problems due to ADM may increase or decrease. The costs would change accordingly, even if there were no change in the population at risk, because a different share is affected.

A further complicating factor is that the causal relationship between ADM and costly consequences may change. Apparent causal relationships might change either because there are new data or analyses. Improved study techniques may simply yield more reliable estimates of causal factors. Additionally, ADM might actually have a change in causal relationship with consequences.

Finally, society's response to ADM may impact on any of these items. Social responses may serve to increase some cost components (where these costs reflect intervention efforts) and reduce other cost components (the result of successful interventions being to reduce certain undesirable impacts of ADM). There are many other ways in which the economic cost to society of ADM might be affected by public interventions. A much debated example of this is the current proposal to make 21 the legal drinking age in all states. Such a change could have immediate cost impacts. The criminal justice system may bear higher costs in enforcing the laws, while there may be lower indirect costs due to reduced alcohol abuse-related trauma.

In updating cost estimates, it is recommended that only two of the six factors be adjusted--wage and price inflation and change in the population at risk.

Prices and population changes can be routinely adjusted because quality data are available on a very timely basis for these continuously changing factors.

The other four factors--incidence and prevalence rates, causal factors, sociodemographic distributions, and social responses--are less likely to change significantly in a short time period than prices, wages, and population. Incidence and prevalence rates for the ADM disorders are not reestimated on an annual basis. It is recommended that detailed cost computations be made when the data from the periodic studies of alcohol and drug abuse become available. The causal relationships of ADM to their consequences are the subject of much scholarly study. New studies on causal relationships will tend to use different data sets, different time periods, and different analysis techniques. These factors make routine adjustments to causal factors unadvisable to undertake.

The impact of new interventions vis-à-vis the economic cost estimates is seldom well monitored, and it is very difficult to integrate the impact of changing social responses to ADM into the cost estimation framework.

C. A Simplified Cost Update Protocol

A two-factor update protocol is recommended. The first factor is an adjustment for inflation, and the second is an adjustment for population growth or real change.

There are somewhat different sets of factors for the various types of cost components. The basic premise of this methodology is that proportional changes (over a period of time) in the adjustment factors are related to proportional changes in the values of the cost components. The period of time is the time between the base year (year with the most recent estimate for the cost component, referred to as "b.year") and the update year (year for which updated estimates for the cost component are desired, referred to as "u.year").

The general update formula is

$$CC_{u.year} = CC_{b.year} \times (AFI_{u.year} \div AFI_{b.year}) \times (AFRC_{u.year} \div AFRC_{b.year})$$

where

$CC_{u.year}$ = value of cost component (CC) in update year (u.year)

$CC_{b.year}$ = value of cost component (CC) in base year (b.year)

$AFI_{u.year}$ = value of adjustment factor for inflation (AFI) in update year (u.year)

$AFI_{b.year}$ = value of adjustment factor for inflation (AFI) in base year (b.year)

$AFRC_{u.year}$ = value of adjustment factor for real change (AFRC) in update year (u.year)

$AFRC_{b.year}$ = value of adjustment factor for real change (AFRC) in base year (b.year).

This formula applies proportional change adjustments for inflation ($AFI_{u.year} \div AFI_{b.year}$) and real change ($AFRC_{u.year} \div AFRC_{b.year}$) to the value of the cost component in the base year to produce a value for the update year.

Table G-1 lists the cost components for ADM and specifies an adjustment factor to reflect change in inflation and a factor to reflect real change. The adjustment factors are in each case a data series that is routinely maintained and published by a government agency. Values for these data series are readily available from publications of these agencies (listed in the table) or by personal communication. The complete references for the publications and identification of the responsible agencies are on the bottom of the table.

Separate adjustment factors are specified for the major components of the costs of ADM. Different factors allow the various cost components to change at somewhat different rates, as dictated by the factors "more" appropriate for each component.

For a given cost component, the same factors are suggested for adjusting estimates for each of the three ADM disorders. The two factors for each cost component reflect general influences that are likely to affect costs of the ADM disorders in similar, if not identical, ways. As stated above, there are many other factors likely to be relevant to cost changes; however, incorporating many or all of those changes in updated estimates would require recalculating the costs from the start. This update protocol is only intended to reflect major, relatively unambiguous, changes that occur over a period of several years. Beyond three to four years, the full cost estimates should be performed again, with an assessment made of new findings and data about ADM.

To facilitate implementation of the update protocol, historical values are presented for the recommended adjustment factors (see table G-2). Data

Table G-1

Adjustment Factors for Updating ADM Economic Costs to Society Estimates

Cost Component	Adjustment Factor	Data Series	Source
Treatment and Support	Inflation & Real Change	Total National Health Expenditures	Health Care Financing Review
Mortality	Inflation	Compensation per hour in the business sector	Monthly Labor Review
	Real Change	Total deaths	
Morbidity-Reduced Productivity and Lost Employment	Inflation	Compensation per hour in the business sector	Monthly Labor Review
	Real Change	U.S. Civilian Labor Force	Monthly Labor Review
Motor Vehicle Crashes -Direct Costs	Inflation	Implicit price deflator for gross national product	Survey of Current Business
	Real Change	Motor vehicle accidents (on the road)	Accident Facts
Crime	Inflation	Implicit price deflators for gross national product: Government purchases of goods and services (state and local)	Survey of Current Business
	Real Change	Crime Index: Number of known offenses	Crime in the United States; Uniform Crime Reports

Cost Component	Adjustment Factor	Data Series	Source
Social Welfare Programs	Inflation	Implicit priced deflators for gross national product: Government purchases of goods and services (state and local)	Monthly Labor Review
	Real Change	Adult population (18-64 years) (as of July 1, each year)	Current Population Reports, Series P-25
Other Direct Costs	Inflation	Implicit price deflators for gross national product	Survey of Current Business
	Real Change	Adult population (18-64 years) (as of July 1, each year)	Current Population Reports, Series P-25
Victims of Crime	Inflation	Compensation per hour in the business sector	Monthly Labor Review
	Real Change	Crime Index: Number of known offenses	Crime in the United States: Uniform Crime Reports
Crime Careers (Drug Abuse only)	Inflation	Compensation per hour in the business section	Monthly Labor Review
	Real Change	Heroin addicts in the United States	National Institute on Drug Abuse
Incarceration	Inflation	Compensation per hour in the business sector	Monthly Labor Review
	Real Change	Total population in state and federal prisons	National Prisoner Statistics: Bureau of Justice Statistics Bulletin
Motor Vehicle Crashes -Indirect Costs	Inflation	Compensation per hour in the business sector	Monthly Labor Review
	Real Change	Motor vehicle accidents (on the road)	Accident Facts

Data Sources for Updating Costs

Accident Facts, National Safety Council, Chicago, Ill., annual.

Current Population Reports, Series P-25, Bureau of the Census, U.S. Department of Census.

Federal Bureau of Investigation, *Crime in the United States: Uniform Crime Reports*, U.S. Department of Justice, annual.

Health Care Financing Review, Health Care Financing Administration, U.S. Department of Health and Human Services, periodical.

Monthly Labor Review, Bureau of Labor Statistics, U.S. Department of Labor, monthly.

National Narcotics Information Consumers Committee Report, Drug Enforcement Administration, U.S. Department of Justice, annual.

National Prisoner Statistics, *Bureau of Justice Statistics Bulletin*, Bureau of Justice Statistics, U.S. Department of Justice, periodical.

Survey of Current Business, Bureau of Economic Analysis, U.S. Department of Commerce, monthly.

Vital Statistics of the United States, National Center for Health Statistics, U.S. Department of Health and Human Services, annual.

are reported for 1975 through 1983 (several values are not available from agencies at this time). The historical data will make it easier to acquire the correct values of new factors.

D. An Example of Using the Protocol

The simplified procedure described above has been implemented to estimate values for 1981, 1982, and 1983 based on the detailed 1980 computations. First, a sample computation for a single cost component--Reduced Productivity costs for alcohol abuse -- is worked out.

Recall that the adjustment formula is

$$CC_{u.year} = CC_{b.year} \text{ X } (AFI_{u.year} \div AFI_{b.year}) \text{ X } (AFRC_{u.year} \div AFRC_{b.year}).$$

The base (1980) value for the Reduced Productivity component was $50,575 million. The adjustment factor for inflation for this component is "compensation per hour in the business sector." The value of this index was 131.2 in 1980, 143.9 in 1981, 155.1 in 1982 and 163.1 in 1983. The adjustment factor for real change for this component is "civilian labor force" in the United States. The value was 106,940,000 in 1980, and the other values are in table G-2.

The update values for 1981-1983 come directly from application of the formula

$$CC_{1981} = \$50{,}575 \text{ million X } (143.9 \div 131.2) \text{ X } (108{,}670{,}000 \div 106{,}940{,}000)$$
$$= \$56{,}367 \text{ million}$$

$$CC_{1982} = \$50{,}575 \text{ million X } (155.1 \div 131.2) \text{ X } (110{,}204{,}000 \div 106{,}940{,}000)$$
$$= \$61{,}612 \text{ million}$$

$$CC_{1983} = \$50{,}575 \text{ million X } (163.1 \div 131.2) \text{ X } (111{,}550{,}000 \div 106{,}940{,}000)$$
$$= \$65{,}582 \text{ million}$$

The updated cost values increased 11.5 percent between 1980 and 1981, 9.3 percent between 1981 and 1982, and 6.4 percent between 1982 and 1983 as a result of both wage inflation and labor force growth in each year. The proportional increase in each factor over one year equals the value of the adjustment factor in the update year divided by the value of the adjustment factor in the prior year minus one. Thus, the increase in wages between 1980 and 1981 was 0.097 = (143.9 ÷ 131.2) - 1 or 9.7 percent.

Similar update computations have been performed for all cost components for 1981, 1982, and 1983 using the base values for 1980 (see table I-1 or

Table G-2

Historical Data Series of Adjustment Factors for Update Protocol

Year	Total National Health Expenditures ($ in billions)	Compensation per Hour in the the Business Sector (Index)	Implicit Price Deflator for State & Local Government Purchases of Goods and Services (Index)	Implicit Price Deflator for for Gross National Product (Index)	Adult Population (18-64 years) (as of July 1) (thousand)	Total Deaths (thousand)	U.S. Civilian Labor Force (thousand)	Crime Index Number of Known Offenses (thousand)	Motor Vehicle Accidents (millions)	Heroin Addicts (thousand)	Population in State and Federal Prisons
1975	$132.7	85.5	129.4	125.8	125,604	1,893	93,775	11,257	16.5	550	N.A.
1976	149.7	92.9	138.3	132.3	128,034	1,909	96,158	11,305	16.8	500	N.A.
1977	169.2	100.0	148.4	140.1	130,407	1,900	99,009	10,936	17.6	495	300,024
1978	189.3	108.6	159.7	150.4	132,020	1,928	102,251	11,141	18.3	470	307,276
1979	215.0	118.7	173.7	163.4	135,329	1,914	104,962	12,153	18.1	420	314,457
1980	249.0	131.2	191.6	178.6	137,842	1,990	106,940	13,295	17.9	492	329,821
1981	286.6	143.9	208.1	195.5	140,054	1,987[a]	108,670	13,290	18.0	492[b]	369,930
1982	$322.4	155.1	222.9	206.9	142,149	1,986[a]	110,204	12,857	18.1	492[b]	414,362
1983	$333.5[c]	163.1	236.6	215.6	144,022	2,010[a]	111,550	(11,960)[d]	N.A.	492[b]	430,830

[a] Preliminary.

[b] 1980 is the most recent year for this estimate from NNICC.

[c] This value has been estimated by RTI based on inflation in personal health care prices and growth in total population. The 1983 value will be available in August, 1984.

[d] Preliminary tabulations as of April, 1984 indicated a 7 percent drop between 1982 and 1983. This translates into a Crime Index of about 11,960,000 known offenses.

N.A.: not available.

III-1), and the available data on adjustment factors. The results appear in tables G-3, G-4, and G-5.

Using this procedure, the economic costs to society of ADM are estimated to increase from $190 billion in 1980 to $213 billion in 1981, $234 billion in 1982, and $249 billion in 1983. The estimates for alcohol abuse, drug abuse, and mental illness do not increase proportionally with the total over the three years of updating because growth in components is different.

The growth in estimated total costs of ADM was greater than the overall rate of inflation in the economy. These estimates indicate growth of 12.1 percent between 1980 and 1981, 9.9 percent between 1981 and 1982, and 6.4 percent between 1982 and 1983. In contrast, inflation as measured by the "implicit price deflator for gross national product" was 9.5 percent, 5.8 percent, and 4.2 percent respectively. The difference between overall inflation and the increase in cost estimated is due to population growth, other real changes, and increased productivity of the work force as reflected by the "compensation" adjustment.

E. A Final Cautionary Note

Updated cost estimates allow the impact of ADM to be roughly assessed in more current dollars and units. The protocol uses the readily made adjustments for the most fundamental changes. While these estimates will be used in place of current cost estimates (or because data for current estimates are unavailable), they are only a substitute for current data. It is recommended that updated values only be used for, at most, three to four years, until it becomes propitious to make completely new estimates due to an accumulation of more recent data, and findings about the incidence, prevalence, and consequences of ADM in our society.

Table G-3

Updated Costs to Society of Alcohol Abuse, Drug Abuse, and Mental Illness, 1981
(dollars in millions)

	Alcohol Abuse	Drug Abuse	Mental Illness	Total
Core Costs				
Direct				
Treatment and Support	$12,052	$1,661	$27,115	$ 40,828
Indirect				
Mortality[a]	15,832	2,168	7,881	25,880
Reduced Productivity	56,367	28,661	3,480	88,508
Lost Employment	4,575	348	20,666	25,589
Other Related Costs				
Direct				
Motor Vehicle Crashes	2,405	[c]	-	2,405
Crime	2,548	6,417	945	9,909
Social Welfare Programs	42	2	222	266
Other	3,239	597	733	4,569
Indirect				
Victims of Crime	189	926	-	1,115
Crime Careers	-	9,570	-	9,570
Incarceration	2,216	1,803	108	4,127
Motor Vehicle Crash (time loss)	512	[c]	-	512
Total	$99,976[b]	$52,154[b]	$61,149[b]	$213,279

Totals may not add due to rounding.

[a] At 6 percent discount rate.

[b] The total costs to society for each of the three ADM disorders are not comparable, since the completeness of data available for each cost category varied significantly. For example, the estimate of reduced productivity is relatively complete for alcohol abuse, only partially complete for drug abuse, and incomplete for mental illness.

[c] Although costs are hypothesized to occur in this category, sufficient data are not available to develop a reliable estimate.

Source: Research Triangle Institute.

Table G-4

Updated Costs to Society of Alcohol Abuse, Drug Abuse, and Mental Illness, 1982
(dollars in millions)

	Alcohol Abuse	Drug Abuse	Mental Illness	Total
Core Costs				
Direct				
Treatment and Support	$13,558	$1,868	$30,502	$45,928
Indirect				
Mortality[a]	17,055	2,336	8,490	27,881
Reduced Productivity	61,612	31,328	3,803	96,744
Lost Employment	5,001	380	22,589	27,970
Other Related Costs				
Direct				
Motor Vehicle Crashes	2,560	[c]	-	2,560
Crime	2,640	6,649	979	10,268
Social Welfare Programs	46	2	241	289
Other	3,479	642	787	4,908
Indirect				
Victims of Crime	197	966	-	1,163
Crime Careers	-	10,314	-	10,314
Incarceration	2,675	2,177	131	4,983
Motor Vehicle Crash (time loss)	555	[c]	-	555
Total	$109,376[b]	$56,563[b]	$67,522[b]	$233,561

Totals may not add due to rounding.

[a] At 6 percent discount rate.

[b] The total costs to society for each of the three ADM disorders are not comparable, since the completeness of data available for each cost category varied significantly. For example, the estimate of reduced productivity is relatively complete for alcohol abuse, only partially complete for drug abuse, and incomplete for mental illness.

[c] Although costs are hypothesized to occur in this category, sufficient data are not available to develop a reliable estimate.

Source: Research Triangle Institute.

Table G-5

Updated Costs to Society of Alcohol Abuse, Drug Abuse, and Mental Illness, 1983
(dollars in millions)

	Alcohol Abuse	Drug Abuse	Mental Illness	Total
Core Costs				
Direct				
Treatment and Support	$ 14,865	$ 2,049	$33,445	$50,359
Indirect				
Mortality[a]	18,151	2,486	9,036	29,673
Reduced Productivity	65,582	33,346	4,048	102,976
Lost Employment	5,323	405	24,044	29,772
Other Related Costs				
Direct				
Motor Vehicle Crashes	2,667	c	-	2,667
Crime	2,607	6,565	966	10,139
Social Welfare Programs	49	3	259	311
Other	3,673	677	831	5,181
Indirect				
Victims of Crime	192	945	-	1,137
Crime Careers	0	10,846	-	10,846
Incarceration	2,979	2,425	146	5,549
Motor Vehicle Crash (time loss)	583	c	-	583
Total	$116,674[b]	$59,747[b]	$72,775[b]	$249,196

Totals may not add due to rounding.

[a] At 6 percent discount rate.

[b] The total costs to society for each of the three ADM disorders are not comparable, since the completeness of data available for each cost category varied significantly. For example, the estimate of reduced productivity is relatively complete for alcohol abuse, only partially complete for drug abuse, and incomplete for mental illness.

[c] Although costs are hypothesized to occur in this category, sufficient data are not available to develop a reliable estimate.

Source: Research Triangle Institute.